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PSYCHIATRY OF OUR COLONIAL FOREFATHERS *

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The records of the reunions of this association are rich in retrospect. It would appear that in our earlier years our members were unacquainted with the custom of presidential addresses. Anticipating what is to be suffered by you in the present instance, those were happier days.

It was not until the 1882 meeting that this feature on the program seemed to have become a fixation. According to the mathematical theory of science, of which the Pythagorean numerology was an early representative and which still lingers in many a mutilated fragment, the figure 8 has great significance. It signified rebirth, and quite according to form, William A. Hammond functioned. Without him there would have been no birth of the American Neurological Association, and with him there came into being that mystic rejuvenation process, the presidential address.

After collating, reading and pondering over the motley menus of previous occasions, I have wondered what might be possible on the way from hors d'oeuvres to dessert that could tempt your attention. There has been such a rich assortment, from pleasing introductions to solid scholarly addresses, that in seeking for some novelty with which to gain for a brief moment your indulgence I have sought an escape in a fantasy construction, the bare outlining of which is all that I can persuade myself to believe can claim your interest.

Fantastic as it may seem, I would offer at this time but a momentary glimpse of an attempt at a piecing together of the "Psychiatry of Our Colonial Forefathers."

I shall ask you to consider for the present only my dream of this progressive synthesis, not yet—almost into the half of the twentieth century—a corpus of data well enough knit into anything but a Joseph's coat of many colors.

Should I ask you to turn in your fancy to a musical metaphor, I would invoke the fire motif of Wagner in "Die Walküre," and in that same mind's eye conceive of the early springing into tiny jets of flame of the spirit that cradled the Colonists. Our more explicit story begins about 1650, a date which stabilizes in a general way the English influence

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which had become most firmly settled along the middle fringe of the eastern shores, with France in the north, France and Spain in the south, and the vast hinterland of the Indian, to say nothing of the Spaniard to the far south of South America and the southwest. Grant the motif to have waxed and waned and then risen higher and higher, until by the Revolution of 1774 our Brunhilde was finally awakened into an organic unity, and ten years later our forefathers became a united people, preeminently English in general culture, still encompassed by an encircling France and by steadily receding Spanish influences.

At this time only the seventeenth century will be under review. At a future date I may attempt something similar for the eighteenth century as a contribution to the history of the development of psychiatry in North America.

In this fragmentary statement I shall spare you the backgrounds of the fifteenth and sixteenth centuries so far as the historical details are concerned. It may become essential to enter into these periods for the picking out of the factors dealing with the mental pabulum that is the burden of this study. Indeed, I shall hope to show that our colonial forefathers were not unacquainted with the ideas concerning things of the mind held by Hippocrates, Plato and Aristotle, and their medieval glosses, and in the struggling groups from Cape Cod to Florida, with its Indian infringements and Negro incorporations there was brewed a witch's brew such as has never been subjected previously to the forces of the creative magic of intellectual fire.

THE EARLY COLONISTS

A brief glimpse of the turning pages of the innumerable records of human activities among our early colonists shows what an inconsistent, helter-skelter, almost chaotic groping here and there took place in the frantic searches for economic advantages in the New World of the seventeenth century on which our interest centers. What a panorama presents itself! Spanish influences, which were so important for the rush for gold of the earlier centuries, may be left aside at this time, although of much significance in our story so far as the southern states were concerned and of specific connotation as bearing on the Indian's attitude toward the invaders.

While the solid background was essentially English, there were innumerable variations in the mental images even among those of English stock. There were the French Catholics, Huguenots of the north and south and some midway, the Puritans of New England, with their dissenting dissenters and the Roger Williams and Anne Hutchinsons and above all their anti-Catholic and anti-Anglican attitudes. There were the Cavaliers of Maryland, few of whom, as the Beards remind us, were really cavaliers. The Scotch-Irish came into New

Hampshire and the Carolinas. The Dutch of New York made their mark, which still persists, even though New York developed a very cosmopolitan atmosphere early. There were Dutch, English, Scotch and French Huguenots in New York throughout the latter half of the seventeenth century. The law upheld the Anglican church, but a tenth of the population only were followers. Anticatholicism was marked. One in five of the population of New York State were negro slaves. There were Germans and Quakers in Pennsylvania, Swedes in Delaware and all around the Indian and French fringe, with the Jesuit missionaries.

The greatest diversity of customs, laws, folkways, manners, beliefs and teachings prevailed. Beard [1] wrote:

> All the eighteenth century tourists from the foreign countries were struck by that fact, by the "strange mingling of the uncouth, the totally wild, and the highly civilized and cultured." They were impressed by the charm of the Southern ladies, the number of excellent French books in the libraries of the planters, the elegant plate on the tables of Philadelphia and Boston merchants.
>
> The everlasting curiosity and questioning of the rural Yankee, the bustling enterprise of the ports, the forwardness of the laboring people, and the range of the intellectual interests.
>
> If the travellers saw Jonathan Edwards shaking all New England over the roaring flames of Hell in 1743, they also heard Benjamin Franklin exclaiming that "the first drudgery of settling new colonies being 'pretty well over,' Americans ought to do their part in scientific and philosophic inquiry."
>
> If they discovered any qualities that could be called distinctly American, they likewise found antagonisms of the most pronounced character. "Fire and water," wrote Burnaby, in 1760, after traveling over a thousand miles in the Colonies, "are not more heterogeneous than the different Colonies in North America." The comfort of the free masses in contrast with the awful beggary of Europe, and the sadness of slavery impressed every voyager. "In the course of 1,200 miles," said Burnaby, "I did not see a single object that solicited charity. The condition of the slaves is pitiable; their labor excessively hard, their diet poor and scanty, their treatment cruel and oppressive."

Before leaving this all too hastily directed glance at the human elements of the early Colonial makings of the United States, "to write a true, full and particular history of which," Wells [2] has remarked, "it would have to be written with charity and high spirits as a splendid comedy," attention may be directed to a certain unity in all this diversity.

The more focal features of this unity have been made the subject of much historical comment and in general may be traced, as Beard, Green, Tyler, Andrews, Fiske and others have done, as the emergence as a cultural type of the English Idea—out of what Fiske outlines as the Roman Idea. The Puritan exodus was but an outstanding symptom

1. Beard: The Rise of American Civilization, New York, The Macmillan Company, 1930, vol. 1, pp. 144, 145.

2. Wells, H. G.: Outlines of History, Garden City, Long Island, N. Y., Garden City Publishing Company, p. 850.

that indicated the emergence into a cultural ego of the value of certain primitive Teutonic strivings which first were instrumental in breaking up the Roman Empire as such, then as early as the Albigensian movement in the tenth century began to foment the reformation and the separation from Romanism through Wyclif, Latimer, Luther, Erasmus, Rabelais, finally to proclaim through the heroic figure of Oliver Cromwell the victory of the English tendencies and a distinctive English cultural horizon with which my completed study would deal.

Fiske [3] summarized his masterly chapter on the Roman Idea and the English Idea as follows:

> It was the ideas of Locke and Milton, of Vane and Sidney, that, when transplanted into French soil, produced the violent but salutary Revolution which has given fresh life to the European world. And contemporaneously with all this, the American nation came upon the scene, equipped as no other nation had ever been, for the task of combining sovereignty with liberty, indestructible union of the whole with indestructible life in the parts. The English Idea had thus come to be more than national, it has become imperial. It has come to rule and it has come to stay.

But before passing from the topic of the individuals as racial elements, one thorny situation provokes attention. This concerns the contacts of our early settlers with the Indian and Negro races. To the psychiatrist, eugenically oriented, this was, is and will remain a portentous issue.

Much research still awaits the psychiatrist who would accurately evaluate the influences of the border fringes of the preexisting Indian cultures. The early colonists did not enter a virgin country. There was already a neolithic culture, separated by thousands of years from the invaders already spread throughout the country. The ethnologic, anthropologic and social sexual data concerning the psychologic reaction formations involved in these contacts (chiefly seen in increased prudery), will receive full consideration in my paper.

The Negro environmental factors, even more sinister, also can claim but a passing reference. In 1607, Negroes from Africa began to be brought into the Colonies as slaves. The Spanish had so used them a hundred years previously. In 1619, they were brought to Jamestown, much to the profit of the southern planter, and the Puritan had no objection to their exploitation. By the time of the closing of the nineteenth century there were half a million. In New York one in every six, and in New England one in every fifty were African stock. What the presence of these primitives meant to the cultural life of the colonists, as seen in the light of psychiatry, and which partly came to the surface

3. Fiske, J.: Beginnings of New England, Boston, Houghton Mifflin Company, 1891, p. 48.

in one gigantic blaze in our Civil War, can be touched on here but lightly.

In considering more in detail what miscegenation may have meant for the super-ego of our colonial men and women, and the unconscious homosexual components, particularly in the men of European, Indian and Negro stocks, we enter a territory still turgid with the greatest fanaticism.

Enough in this foreword of brief characterization of the elements that went into the composition of this medley both as to the primary migrations and as to the later reenforcements of emigration and territorial expansions.[4]

THE COLONIAL MIND

What in equally fragmentary a manner shall I present as to the spirit of the bubbling, the general tendencies, as seen from the angle of psychiatry? First let me force a definition of psychiatry as I shall here attempt to deal with it. For us at this time psychiatry will be the philosophy of health in its entirety, individual and social, in the sense that Democritus once wrote,[5] "It is meet for men to take account of the soul rather than of body for perfection of soul corrects wretchedness of the body tabernacle, but bodily strength without reasoning makes the soul not a whit better." Or in the more modern sense I would claim that all of man's organs, even down to his ionic milieu are under the control of his mental states, most of which are operating unconsciously. Psychiatry is the science dealing with mental states, hence a comprehension of the entire behavior of the body is only adequate when the mental states are understood.

In this section of my study I will endeavor to outline the attitudes of these people so far as the unitary-body-mind-problems were concerned. This was dimly outlined in the Heraclitean, Protagorean, Socratic Ideal. Under the influence of Aristotle, it suffered a deep regression.

Medicine as a cultural discipline is now in the rompers of science. In those days it was in the swaddling clothes of theology, and theology in one aspect of scholasticism accomplished the almost complete castration of all that was essentially dynamic in Aristotle.

In another place, in discussing the function of Arabian psychiatry in bringing back to European culture the dynamic conceptions of the presocratic and socratic thinkers, I have quoted Sarton, who has most pertinently said that the darkness of the Middle Ages was never as great

4. Greene: Provincial America, New York, Harper & Brothers.

5. Democritus, in Diel: Die Fragmente der Vorsokratiker, no. 187, Berlin, 1922, vol. 2, p. 99; quoted from Scoon: Greek Philosophy Before Plato, Princeton, N. J., Princeton University Press, 1928, p. 221.

as our ignorance of them. So, in my thesis I have chosen to depart from the usual condemnation of the "Dark Ages." In fact, I find a continuum of evolutionary advance, which interestingly enough is more consistent through the medical phylum than almost any other province of the sciences, consistent may I add as to the retention of the superstitions of the past as well as to the slow advance into the future. In fact, the idea of Democritus that the "boundary between the useful and the injurious is pleasure and pain" has been the place where the medical discipline has always focused its interest, in the battle between "health and disease."

In spite of certain readily evaluated prejudices, Draper's great classic on the "Conflict Between Religion and Science" gives a valuable close-up for our special issues, even more generously illuminated by Lange in his "History of Materialism." If psychiatry may claim a certain prestige as a large generalization which would combine in one all-embracing effort an understanding not only of the part played by structure in disturbing individual adjustments but also of how ethical conflicts, within the individual, as also within groups, bring about disease, then it may be stated that a resolution of these conflicts is imminent as psychiatry presses forward in its scientific analysis of the function of ethics and in its survey of the evolution of the control of human behavior from magic to a scientific ethics.

The Middle Ages were prolific in the development of intuitive religious emergents. The outlining of the history of scholasticism can well be left to Lange, with a reminder from Thorndike,[6] who, quite in sympathy with Sarton and a thought already hinted at wrote:

> The old slurs and disparaging generalizations at the expense of the Middle Ages are now repeated only by mechanical creatures of habit, by those who stopped thinking and reading twenty to thirty years ago, and who refuse to give up any catchword or prejudice that was instilled into their minds in childhood.

Side by side the Geisteswissenschaften were establishing contacts with these intuitively grasped inner experiences. At the time of our colonial consolidation, biology and religion were at the great beginning of the establishment of a new cultural horizon which more recent historical research would show had its first impulses in the twelfth century.

May I ask your attention to a formulation of an interpretation of culture, not entirely original but put in my own way, to me at least intriguing? This generalization would follow the pattern of the biologic sciences, and I have used the term paleopsychology as an analog of paleobotany and paleozoology. With this conception in mind, then, our understanding of a culture would follow the earlier formulations of a

6. Thorndike, L.: Science, Thought in the Fifteenth Century, New York, Columbia University Press, 1929, p. 9.

geologic culture. A paleozoologic horizon is considered as higher or lower in terms of the presence of evolutionary emergents of a more advanced type. Thus Ordovician is characterized by the presence chiefly of fishes—as reptiles emerged the horizon advanced. So, in the mental sciences, the colonial period under consideration is not to be measured culturally in terms of its possible African paleolithic or Indian neolithic accidental incorporations—some of the features of which were, are and will be of great significance for psychiatry—nor in its abundant inclusion of paleopsychologic magic fossils such as the King's touch, the belief in witches, not only at the European level, but also with Indian and Negro contaminations at a deeper level, the crude polypharmacies, autistic thinking, of devils and doctors, but through the recognition of the presence in the cultural horizon of the revived thoughts of the Greek dynamic thinkers, and the oncoming wave of novelties and borrowings through Francis Bacon, Descartes, Giordano Bruno, Spinoza, Leibnitz, Locke, Hobbes, Hume and Voltaire, Melanchthon, Luther and Calvin, Linacre, Rabelais, Paracelsus, Leonardo, Vesalius, Sylvius and—but I spare you the pedantic recital of more of the great thinkers of the Renaissancé whose entry into the cultural horizon just preceded or were coincident with the birth of the elements of our colonists.

A BEHAVIORISTIC PSYCHIATRY

Psychiatry seen from the angle of behavior may well be claimed as the oldest of the sciences. Why men did things has been debated since the beginning. It matters little that the forms taken by the interpretations have altered from magic to science, for with some understanding of the process of rationalization and of symbolization the substance of reality may be seen behind the greatest diversity of the rationalized forms, going back to the most ancient of myths. It is of comparatively small importance that the names given to the "unconscious" have changed through the ages. The work that it does has been felt from the beginning even if not rationally verbalized. "It is with our entire past that we desire, will, and act." "Our past is manifest in the form of impulse." "Whereas only a small part is known in the form of idea." These are but fragments of Bergson's sweeping thought.

The story tellers of Genesis have preserved for us the myth of Noah's Ark and the flood. The unconscious realities hidden behind the symbolizations, distortions and dramatizations reveal the simple story of the genesis of every living thing; "contact, impregnation, gestation, and birth."

The period of our colonial growth was one of vivid awakening. In our Wagnerian fire motive, the flames of newer, wider and deeper understandings were leaping up rapidly all over Europe and the religio-

philosophic forms of verbalization were breaking up before the analytic experimental methods.

Silberer, among others, has shown that the more or less widespread notion that alchemy, with its mystic envelop, was simply the materialistic search for transformation of baser metals to gold, is quite false. And in a similar frame of reference it may be defended that there was much more good psychiatry behind the religiophilosophic verbal forms of the latter half of the seventeenth century than a too ready putting of present day meanings into the phrases of those earlier days would seem to imply. The "Devil" after all was a pretty broad synthesis, even in those days, and only for folk medicine and the ignorant did it mean what more of us mean by it today. There are at the present day millions, and not all in Tennessee, whose paleopsychologic horizons are still filled with the thought fossil forms of medievalism, and still more millions whose "Devil" has but shifted a few points higher in the cultural scale.

In my thesis I try to indicate the details that lie behind the thoughts of Linacre, of Bacon, of Vesalius, of Harvey, of Shakespeare, of Descartes, of Locke and others which were far removed from these folk medicine superstitions. Even though these thinkers stood out as isolated peaks against the low plains of ignorant chatter, it is through such super-ego images that the psychiatry of the times must be judged rather than by the persistent remnants of invested thinking which behave as a social compulsion neurosis.

Indeed there is a definite grim, if not sardonic, humor, in the fact that the one state in which the first principles of freedom of religion, politics and thought were implanted by Roger Williams should have been and still remains the smallest in this great country of ours. It was in the year 1654, eighteen years after his founding of what was to be Rhode Island, that we find Williams writing to Sir Henry Vane:[7]

> We have not felt the new chains of the Presbyterian tyrants, nor in this colony have we been consumed with the overzealous fire of the (so-called) godly Christian Magistrates—Sir, we have not known what an excise means; we have almost forgotten what tithes are, yea, or taxes either to church or commonwealth.

Intense religious preoccupation has always had an unmistakable psychotic coloring.

It would require a wide survey to present even the faintest glimmerings of the intense hatreds that glowered, sparkled and burned with all the luxury of the virgin timber among the early Puritans. Our Comstocks, Watch and Ward Societies and fundamentalists are but a few

7. Easton, Emily: Roger Williams: Prophet and Pioneer, Boston, Houghton Mifflin Company, 1930.

of the persisting tendencies of the Jehovah complex that played so large a rôle in the early mental life of the colonists.

This widespread trend for identification with God and wholesale knowledge of the wishes of Providence [8] can be thoroughly set forth only by much detailed evidence. Suffice it to say that, in spite of the early rigors of the first quarter century, nature was an abundant provider for those who had any industry at all. Man in his conceit has always thumped himself on the chest and given himself the benefit of Providence in such situations. This is one factor that has built up the peculiar Elmer Gantrys of this country. It is also noteworthy that behind all this pioneering, adventurousness, eagerness, coupled with all the superpiety, the erotic life was greatly in need of extrapolicing. This was due not only to the comparative scarcity of women, which put a large stress and strain on the homosexual jealousy component, but to the presence of inferior races, comparatively free from the sexual taboos of Europe; the latter factor was even more important than the former in fostering the extreme mental reaction formations of religious bigotry, the equivalents of many present day psychotic pictures, in the early colonial days.

Psychoanalytic study has shown that the old saying "honi soit qui mal y pense," which originated as a flash of intuitive cynicism, is really a sound bit of understanding. The Comstocks of the world are after all dirty fellows, and their prototypes were abundant in the colonies.

In those early days there was much pious window dressing of sober cloaks and plain linen, demure countenances and measured speech. In Massachusetts even the General Court busied itself with forbidding the sale and purchase of garments trimmed with lace. I suspect that Calvin was more read than Shakespeare. Certainly Johnson, Bacon, Cervantes, Beaumont and Fletcher were of the Devil.

We have some traces of the elder Winthrop's library. We are told there were little brown books on magic from Frankfurt, Latin and even German tomes on alchemy, the Philosopher's Stone, astrology and the various mysteries. "Mr. Winthrop kept a journal" in which "an admixture of cramped sexual curiosity added a sinister element." "In it, side by side with affairs of state, stood details of adulteries, sexual perversions, and obstetrical misadventures occurring in the Bay. The salty Elizabethan had become the pornographic Puritan." [9]

Superstition has its roots in and derives its power from the "worship of Baal and Ashtaroth," and only a complete understanding of the significance of the story of Lot will open our minds to the rampant

8. Augur, Helen: An American Jezebel, in The Life of Anne Hutchinson, New York, Brentano's, 1930, p. 69.

9. Augur, Helen (footnote 8, p. 68).

sadism of our colonial forebears, an inheritance from the general European background of the religious struggles of the reformation and inquisition, with the special new environmental features which surcharged the ambivalents so that license threatened liberty, and freedom could be strangled by intolerance.

Luther [10] was not free from hypochondriacal fears and depressions. While in his retreat on the Wartburg, we read that he suffered from severe constipation attributed by him to devils. Melanchthon was even more superstitious, and told many ghost stories. Jean Wier (born 1515) has given us a rich collection of these early devil believers. He was a prototype of our modern exposers of psychic medium frauds.[11]

One needs not be reminded that George Fox, the founder of Quakerism, suffered severe mental confusion as he ran up and down the streets of Lichfield barefooted, crying out—for the frozen streets seemed to run blood—"Woe to the bloody city." This was in 1651, and at the same time that Bunyan also was in turgid mental waters. Both were manic-depressive. Bunyan's "Grace Abounding" shows many evidences of his depressions, even taking into due consideration the religious formulas of the times.

The troublous cromwellian times made the cradling of most of our Puritans. O'Donoghue,[12] in his fascinating Story of Bethlehem Hospital, tells how Daniel, Cromwell's porter, was admitted to Bethlehem in 1656—and notes how his master pooh-poohed the grandiose rhetoric of his former servant as the emanation of a crazy brain of a man who had been "so many years locked up in a little cell with no other furniture but the torn leaves of three or four Bibles"—and the imperturbable Daniel's reply is on record: "I was indeed mad." "For that matter every mortal man is more or less mad, when he is in love, for example, when he becomes a miser, but, most of all, when he is ambitious. Your madness, however, was worse than mine, for you set the world on fire. Men bring even to this place (Bethlehem) some germs of their former madness, and the truth is—between friend and friend, you know—that you are very far gone, and you must take a course of Lethe waters for six months at least."

This was all while Bethlehem was in its old home and while not so many years previously Dr. Crooke enjoyed one of those ever perennial "investigations," when Burton (1576-1639), when not immersed in his books in Christ College, Oxford, actually paid visits to the hospital, for he mentions it three or four times in his "Anatomy of Melancholy"

10. Luther: Sammtliche Schriften, Halle, 1743.

11. Wier, J.: De praestigus daemonus et incantionibus et veneficus, Basle, 1563; see French translation.

12. O'Donoghue, E. G.: Story of Bethlehem Hospital, London, T. F. Unwin, 1914, p. 187.

(1621); this book, a rare mine of historical summary, was probably on the shelves of Meverall, Nurse, Tyson and later of the Monros, and I suspect was probably brought to this country early, although when and where I have as yet been unable to trace. "Music, mirth and merry company," says Burton, are sovereign remedies against melancholy, which with much of the context of this remarkable work gives evidence of more reading of the classics than actual work among the patients.

Should one know more fully the ideas current among our Puritans about madness, I suspect that much more will have been gained from the works of Nicholas Breton (1545-1626). In his "Forte of Fancie" (1584) may be found many a trace of his visits to "Bethlehem." As O'Donoghue remarked, his anatomy of melancholy was not written out of ancient tomes, after the manner of Burton. Thomas Dekker (1577-1638) has laid whole scenes in the hospital—of which more in my paper. Shakespeare, who had been associated with Bishopsgate by residence and by friendships, distilled the noxious philtres which he administered to Ophelia and Hamlet, from the herbs of Bethlehem's garden.[13] The fire of London (1666), be it remarked, spared this garden.

Literature will give a far better picture of the ideas of our colonial forefathers than any of the more musty tomes which I am compelled to mention. I cannot dwell at length on the Shakespearean ideas of psychiatry in this flickering candlelight thrown on my study, but Shakespeare, like Sophocles, has shown the grasp of genius as to the real significance of the relation of the forces of the thwartings of the instinctual life to the mental defense phantasmagoria with which psychiatry would deal. A chapter on the Shakespearean predecessors, Beaumont and Fletcher, Johnson, Middleton and the craftsmen of the mystery and miracle plays—"Everyman" and others—show how virile was the mental awakening at the time of our colonization. Bedlam moved to Moorfields in 1676; Pepys and Evelyn in their diaries had much to say of it, and it was here in the incurable ward, fifty or more years later, that Hogarth painted his remarkable painting of the Rake's Progress (1732-1733-1735).[14]

Though it may be held and more or less proved that a better cross-section of a cultural horizon, even for psychiatry, may be obtained through a careful study of the general literature, the records of the counting house and the courts, to neglect entirely the technical medical literature would be one-sided.

At the present time, it is all too well recognized how quickly a textbook gets out of date. One finds a similar thing as one even cursorily dips into the literature of medicine of the sixteenth and

13. O'Donoghue (footnote 12, p. 145).

14. O'Donoghue (footnote 12, p. 245).

seventeenth centuries. Our chosen field was not neglected. Though only a few names stand out in the sixteenth century, some are of outstanding preeminence, even though contributing but fragments to our crib. Linacre, Rabelais, Paracelsus, Leonardo da Vinci and Vesalius, among the larger lights; Montanus, Fernel, Trincavalla, Rondelet, Nicholaus Piso, and finally Felix Platter, Prosper Alpinus and Jean Wier, among the lesser.

One can admire even today the thoughts of Wier [15] in his onslaught on the rampant demonology of the times, less hidden then than is now perceivable in the more allegoric Rabelaisian stories. His attack on the fundamentalists is almost as rich as Mencken's "Americana." The light thrown by his work on the hysterical phenomena is not to be lightly brushed aside.

I cannot stop to examine in detail the thoughts of either Wier or Platter, or present a digest of the theses dealing with mental phenomena which appeared in the century of Vesalius or Leonardo, and which were available to the outstanding thinkers of the century of our colonization. Laehr catalogs at least 800 volumes published during the sixteenth century, dealing with one or another phase of mental medicine. About one half are reprintings of either the older classics or new editions of the classics of the century; of the remaining 300 or 400, Friedreich, in the only complete history of psychiatry published just one hundred years ago, refers to only about thirty.

Nor can it be said that the strictly medical writers of the seventeenth century contributed much original material. Neither shall I clutter the recital of this prolegomena with the names of those more fully dwelt on in this study. Certainly for the first half of the seventeenth century there is little evidence as yet as to what books were read by the early settlers. Reading began early with us, however, and a colonial epic could be written tracing the evolution of reading lamps from the time of whaler's blubber drip—to Pratts' astral oil. What is most striking is the beginning of the use of local idiom in the last quarter of the century. Practically all of the schools of Greek thought had their adherents; Aristotelians predominated; there were Galenists of varied hues. The Bible was the only universal textbook on psychiatry. The King James translation came in 1611.

The humors are commencing to be pressed by devils, however, and, what is more significant, by their more ethical abstractions, namely, the influence of "sin" in the causation of mental disorders. Although syphilis was recorded in Massachusetts by Winthrop as early as 1646, little was known of its mental concomitants.

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15. Wier, Jean: Bibliothèques diaboliques, Paris, Delahaye & Secrosmer, 1885, vols. 1 and 2; available in French.

RELIGIOUS CULTS

Our problem here is very intriguing. This concerns the foundations of our amazingly rich crop of religious cults, richer than anywhere else and comparable with what happened in the Mediterranean basin in the bronze age, where, if we would see eye to eye with Elliot Smith, a somewhat similar witches' brew took place. The admixture of many racial strains of closely related cultural horizons with those of the Indian and Negroid levels, with their consequent sexual taboos and multitudinous religious reaction formations, is responsible for this amazing medley; it was most strikingly focalized in later years in our far southwest, where Indian, Negro and Spanish miscegenation had been going on for a few antecedent centuries.

A real understanding of the religious life of our colonists and its later phases can, I believe, come only from the psychiatrist, who will be able to trace the evolutions of the Oedipus situation as influenced by the Indian and Negroid level contacts.

This thought might seem irrelevant, save as one sees that ethical situations enter more distinctly into etiology in the minds of the physicians of the colonial times. The case of Mary Dyer of Boston, who gave birth to a monstrosity and was later hanged (1660) by Quakers who considered (in their unconscious at least) that she had had intercourse with the devil, for the monstrosity had horns (Oedipus situation). The crude "devils" of medievalism begin to be submerged very slowly, and the early glimmerings of mental dynamics in prototypes of the ego, super-ego and id appear.

Thus for certain authors, the self-accusations of the manic-depressive cases commence to be seen in a different light. Fabricius (1560-1634) notes the occurrence of the manic-depressive cycle. This observation gives Friedreich (1829) the occasion to comment on somatic disease alternates for psychic disorders. While Horst (1578-1636) preaches humors, Sennert (1572-1637) dwells on demons and seems to have known of Horace's indispositions.[16] Sennert seems to have observed in deliria, etc., the return of unconscious material, such as foreign languages, etc., and ponders over it. It is also said of Sennert [16] that he was among the first to doubt the value of tickling the neck or the clitoris for hysteria as indicated by Galen and advised by Avicenna—apparently, however, more on "Christian" grounds. Zacutus Lusitanus (1575-1643) reported unique modes of dealing with fixed ideas—delusions. One patient complained of being so cold that only being put in a burning fire would restore his natural warmth. He was promptly sewed up in a fur sack, and became well when the fur was set on fire. Nicholas Tulp, undoubtedly known to the physicians of New

16. Dubois, F.: Historie philosophique de l'hypochondrie, Paris, 1833, p. 480.

Amsterdam, reported an interesting case of catatonic perseveration in which the patient continually beat on his knee with his fist. He names the condition "malleatio." Rembrandt's portrait of him is remembered better than his medical "Observationes Medicae" in which the first descriptions of a manlike ape are to be found.[17]

Of Thomas Willis (1622-1675), who stands out in the century, I can say now only that one finds in his writings the first intimations of the dementia paralytica picture. Sydenham, late in the century, was the most widely read medical author.

Our early colonial physicians, at first mixtures of preachers and doctors, were all educated in Europe. Aberdeen, Oxford, Edinburgh, Cambridge, Amsterdam, Leyden, Utrecht, Paris, Padua, and London were the chief universities from which they came. Although Harvard College was founded in 1638, it was not until a hundred and fifty years later, 1781, that Doctors of Medicine were graduated from it. Green[18] says that until 1750 only nine graduates of Harvard took medical degrees, two from Padua, one each at Cambridge, Oxford, Aberdeen and Leyden, and three others not known. No medical journal was found until late in the eighteenth century (*Medical Repository*, 1798).

As the various doctors of this period pass by "dim-panelled in the painted scene of sleep," I catch for a moment the figure of one, and, in the words of Hawthorne in "The Scarlet Letter," hear the following dialog:

> The disorder is a strange one; not so much in itself, nor as outwardly manifested, in so far, at least as the symptoms have been laid open to my observation. Looking daily at you, my good sir, and watching the tokens of your aspect now for months gone by, I should deem you a man sore sick, it may be, yet not so sick, but that an instructed and watchful physician might well hope to cure you. But I know not what to say, the disease is what I seem to know, yet know it not.
>
> "You speak in riddles, learned sir," said the pale minister, glancing aside out of the window.
>
> "Then to speak more plainly," continued the physician, "and I crave pardon, sir, should it seem to require pardon, for this needful plainness of my speech. Let me ask as your friend, as one having charge, under Providence, of your life and physical well being, hath all the operations of this disorder been fairly laid open and recounted to me?"
>
> "How can you question it?" asked the minister. "Surely it were child's play to call in a physician and then hide the sore."
>
> "You may tell me, then, that I know all?" said Roger Chillingworth, deliberately, and fixing an eye, bright with intense and concentrated intelligence on the minister's face. "Be it so! But again! He to whom only the outward and physical evil is laid open, knoweth, often times, but half the evil which he is called upon to

17. Jones, F. W.: Man's Place Among the Animals, New York, Longmans, Green & Company, 1929, p. 47.

18. Green, S. A.: History of Medicine in Massachusetts, Boston, A. Williams & Company, 1881.

cure. A bodily disease, which we look upon as whole and entire within itself, may after all, be but a symptom of some ailment in the spiritual part. Your pardon once again, good sir, if my speech give the shadow of offence. You sir, of all men whom I have known, are he whose body is the closest conjoined and imbued, and identified, so to speak, with the spirit whereof it is the instrument."

"Then I need ask no further," said the clergyman, somewhat hastily rising from his chair. "You deal not, I take it, in medicine for the soul?"

"Thus, a sickness," continued Roger Chillingworth, going on, in an unaltered tone, without heeding the interruption, but standing up and confronting the emaciated and white-cheeked minister, with his low, dark, and misshapen figure, "a sickness, a sore place, if we may so call it, in your spirit hath immediately its appropriate manifestation in your bodily frame. Would you, therefore, that your physician heal the bodily evil? How may this be unless you first lay open to him the wound or trouble in your soul?"

"No, not to thee! not to an earthly physician."

Are we in the twentieth century yet willing to invert the old saying "mens sana" into its more fully evolved form "corpus sanum in mente sana" thus so ably outlined for the days of our colonial ancestors?

Dreams are chiefly characterized by their disjointed structure and by their abrupt endings. And thus, true to form, I awake at this point from my reverie. In asking you to deal kindly with this dream fragment, I feel assured of the continuance of that chivalry of spirit that animated you in placing me in this position of honor and which has given me so much encouragement and has contributed so richly to my happiness in this enterprise.

64 West Fifty-Sixth Street.

THE CASE OF WILLIAM COWPER, THE ENGLISH POET *

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When some of us older members of this society began to study mental diseases, we had a very simple classification. There were mania, melancholia, monomania and dementia, as the four main groups. Additions and modifications were made in due time, and names were changed. The toxic and infectious psychoses and those caused by organic lesions of the brain, such as dementia paralytica, came to be more fully recognized. Then, of course, there were the psychoneuroses, such as hysteria and neurasthenia; finally, there was the great group of the idiocies and imbecilities. The merits of this scheme were its simplicity and that it was in accord with facts.

When I say that it was in accord with facts I refer especially to the distinction which it drew between mania and melancholia. These two were separate diseases, the antitheses of each other. No one in those days would have thought of denying to each its own identity. We thought it true, because we thought that it could be demonstrated in every asylum. The maniac had one disease; the patient with the melancholia, another. With the inevitable progress that has overtaken psychiatry, however, there came a change. Kraepelin merged these two entities into one. We have now only manic-depressive insanity (at least in the books). Melancholia itself has met with a melancholy end. A recent writer banishes it to old age. He says that it is only involutional and not to be seen before the patient is 50 years of age.[1]

The case of William Cowper is of interest, not only because of its literary features, but also because it raises this very question of nomenclature or even something more than nomenclature. It raises the question of facts. It is a historic case, the story of a man who was often insane and who nevertheless won a permanent place in English literature.

Cowper was born in 1731, the son of an English clergyman, who had been chaplain to the king. His ancestry presents some interesting points in heredity, and is referred to by Francis Galton, the founder of eugenics.[2] Two of his nearest kinsmen were distinguished judges. The

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1. White, W. A.: Outlines of Psychiatry, ed. 12, Washington, Nervous & Mental Disease Publishing Company, pp. 241, 242.

2. Galton, F.: Hereditary Genius, New York, The Macmillan Company, 1914, pp. 231, 232.

first Earl Cowper, his great uncle, had been Lord High Chancellor of Great Britain, the highest judicial magnate of the kingdom. His grandfather, Sir Spencer Cowper, a younger brother of the Lord Chancellor, was one of the Justices of the Common Pleas. It is a curious fact that this judge, when a young man, had been tried for murder but acquitted for lack of evidence. The alleged victim was a pretty girl, who seems not to have been murdered but to have drowned herself for love of the future judge.

On his mother's side, Cowper was descended from the Donne family, which claimed to trace its lineage back to Henry III, one of the Plantagenet kings. It had produced a poet, John Donne, who was held to be one of the first geniuses of his day, but is now well-nigh forgotten. This heredity of Cowper's on both sides, and the environment which it indicates, are worth remembering as one follows the career of the poet.

Cowper was a hypersensitive child, who suffered from the brutality of his schoolmates. He has left a poem which reflects severely on the public school system of England. He was educated at Westminster, then one of the foremost English schools. The education was purely classic, and Cowper was such a good scholar that he read Homer's "Iliad" and "Odyssey" for amusement. He did not proceed to either of the universities, but on leaving Westminster was apprenticed to a lawyer and later entered the Temple, or law college, and in due time was admitted to the bar. His prospects, because of his family influence, were good, but he seems not to have studied law with any enthusiasm and he mingled with gay and dissipated companions. His first mental breakdown came when he was about 30 years of age, and the circumstances display the weakness of his character.

This weakness is best described as a tendency to vacillation or alternation of mood—a swing from low spirits to high spirits and then back again. It was almost a rhythm or cycle, and it was a feature of great significance in Cowper's case. Its influence can be traced in some of his literary work later in life.

While he was in the Temple he had suffered from dejection of spirits, with religious doubts, but after a holiday in the country this melancholy had given way to such elevation of spirits that he burnt his prayers, an act which afterward added to his remorse.

The immediate cause of his first serious attack was his appointment, through family influence, to a lucrative clerkship in the House of Lords. This would have required careful preparation and passing an examination before the peers. The prospect so unnerved Cowper, who had worked for months to prepare himself for it, that as the day approached he fell into extreme agitation. His terror was mingled with religious

doubts, which returned once more to plague him. He has left a memoir, written in his own hand, of his mental state at this time, a voluminous and rather verbose document, filled with a farrago of religious despair.[3] He also developed delusions of suspicion, and never went into the street but that he thought people stared and laughed at him and held him in contempt. He read an anonymous letter in a newspaper and believed that it was meant for him. If he opened a book, such as Shakespeare or the Bible, he thought that the first passage he glanced at was meant as a special denunciation of himself. He had frightful dreams, which confirmed his delusions, and he seems to have heard voices. It was at this time that he conceived the delusion of the unpardonable sin, a delusion which was never again to leave him.

In this state of despair he attempted suicide, but his attempts all failed; they even suggest that they were only pantomime. He first bought a bottle of laudanum, smelt it and threw it aside. He then went to the river to throw himself in, but the sight of the water repelled him, and he turned away. He went to bed with the blade of his knife pressed against his heart, but he had not the nerve to thrust it in. Finally, he strung himself up with his garter, but the garter broke just in the nick of time and let him drop to the floor. He had nothing to show for it but a bloodshot eye and a sore place on his neck. Evidently Cowper was not made for suicide.[3a]

His condition was now so bad that he was sent to a private asylum for the insane, kept by a Dr. Cotton, where he remained for eighteen months. During the first half of this period he was in a state of profound melancholia, from which he emerged rather suddenly in a state of great exaltation. This hypomania was indeed so marked that Dr. Cotton evidently feared an access of acute mania, but this did not develop. It was then that Cowper's religious conversion occurred, and was accompanied with great elation of spirits. He was in such a joyous state of mind that he even grudged the hours which he had to give to sleep. It was a "spiritual ecstasy," which continued even after he left Dr. Cotton's care.[4]

This was the period of the widespread religious revival led by Wesley and Whitefield, and Cowper fell so completely under its influence that he has sometimes been called the "poet of a sect." [4a] It was an emotional

3. Southey, R.: Life of Cowper, Boston, Otis, 1839, vol. 1, chapter V.

3a. Memoir of the Early Life of William Cowper, Esq., Written by Himself, Philadelphia, Earle, 1816.

4. Smith, Goldwin: Life of Cowper; English Men of Letters, New York, Harper Brothers, p. 24.

4a. Childs, H.: Cambridge History of English Literature, article on Cowper, vol. 11, p. 89.

evangelical type of religion, which made its appeal directly to the individual, promoting introspection and a sense of sin, followed by conversion. As Goldwin Smith well said, it had its extravagances. They were not of a kind to promote the welfare of a man like Cowper, although they inspired some of his best known verse.

When Cowper left the asylum in this state of joy, his family, probably not knowing what else to do with him for he was totally unfit to take care of himself, secured him a lodging in the small town of Huntingdon, where he became an inmate of the home of the Unwins, an evangelical family of the most approved village type. Their days were spent in a round of religious observances, family prayers, reading the scriptures and sermons and singing hymns. Cowper himself describes this life with unction "as consistent with the utmost cheerfulness." So it was as long as the cheerfulness lasted, which was not very long.

Mr. Unwin died, and Cowper and Mrs. Unwin moved to the village of Olney, where began between them what has been called one of the most noteworthy of literary friendships. They occupied the same house for twenty years, never married, and yet the breath of scandal never assailed them. Mrs. Unwin was a village saint, who coddled the poet in all this domestic intimacy and remained immune. Cowper by that time was evidently not a man who could be suspected of immorality. His relations with his fair housemate were purely platonic. Mrs. Unwin was only a few years his senior, and this queer alliance presents a problem in the psychology of sex. He wrote poems to her, and called her his Mary.[4b]

Another inamorata was Lady Austen, who appeared on the scene later as a visitor. It was said that Cowper wanted to elope with her, but it was probably not true, for Cowper's relations with women all through his life seem to have been those of a child or ward rather than of a man. There is little that is amatory in his poetry.

Olney was a remote, squalid village in a pestilential country on the river Ouse.[5] It had a population of lacemakers, who were ill paid, fever stricken and for the most part as brutal as they were poor. There was not a woman in the village with whom Mrs. Unwin could associate. The house was dismal, prison-like and tumble-down. The place, especially in the winter, must have been repelling. Cowper's only exercise then was in a small garden. The moving spirit in Olney was the Rev. John Newton, an evangelist and religious enthusiast, who preached a kind of mysticism and miracle, sudden conversion, special providence

4b. Hayley, W.: Life and Letters of Cowper, new ed., Chichester, Johnson, 1809, vol. 1, p. 113.

5. Smith (footnote 4, chapter III). Many of these details are taken from this chapter. Southey (footnote 3, vol. 2, pp. 102 and 103).

and satanic visitations. He had a reputation for "preaching people mad," and he doubtless confirmed Cowper in his delusions. Here Cowper spent his days in a round of religious exercises without relaxation or relief. He was Newton's assistant at prayer meetings and visiting the poor, the sick and the dying. Seclusion from a sinful world was a part of this semimonastic system. Cowper was cut off from his friends and from people of his own social class. He dropped his correspondence and sold his library, so that he had but few books.

As a relaxation, the Reverend Newton (who saw too late that this abnormal life was telling on him) induced him to write hymns, in some of which can be seen today the shadow of an advancing madness. He again went insane, fancied he was rejected of heaven, ceased to pray, and again attempted suicide. The Reverend Newton did not at first even call in a physician; he looked on the attack as the work of the devil, and himself treated Cowper for more than a year. We can guess what the "treatment" by such a man for such a patient in such a place must have been. Cowper seems never fully to have recovered from this attack which rendered his life a blank for five or six years. His delusions remained fixed and unalterable.

Cowper's real literary work did not begin until he was about 50 years of age. Mrs. Unwin was the inspiring muse. She had the good sense to induce him to write, for she evidently saw that he needed wholesome occupation. He had thus far written only some detached poems, some of them, however, among his best. He now wrote a series of what he probably intended for moral satires on the world—by a man who had almost forgotten the world. The book fell dead from the press. Next he wrote "The Task," his longest poem. It is poetic and sentimental, but rather commonplace, with its musings on nature and rural life and too much preaching and moralizing. Although the poet calls himself a "stricken deer that left the herd," the poem gives little evidence of his morbid mind, but shows rather a man contented with himself and his own world. It is signficant for that very fact, for Cowper had somehow learned to repress his morbid emotions while he wrote his best verse. He can appear cheerful, even gay. This is an aspect of his case which today should invite to psychoanalysis. The poem succeeded and made him famous.

This elevation of mood in a confirmed melancholic is best shown in his famous poem of John Gilpin's ride. This is one of the best humorous poems in English literature, second perhaps only to Burns' poem of Tam O'Shanter's ride. The story was told to Cowper by Lady Austen, a visitor, who wanted to amuse him. He lay awake at night laughing over it, and in the morning produced the poem of the immortal ride. It had immense success, was recited in London to large audiences and was

sold in thousands of copies. It was written apparently in a hypomanic mood; but so inveterate was Cowper's despondency, that he seems to have taken no great pleasure or pride in its success; and when afterward he was in one of his spells of profound depression he could not bear to hear John Gilpin even mentioned.[6]

From our standpoint, Cowper's most noteworthy literary work was his translation of Homer. It is significant, because it raises the question of diagnosis. He was 53 years of age when he began it, and about 60 when it was first published. This man, who had once sojourned in a mad house, and had lived an abnormal secluded life and given up his classic studies for many years, who believed that he was a lost soul and had twice attempted suicide, settled down when past middle life to a task of scholarship such as is unexampled in the history of the insane. Yet it was while he was engaged on this work that he had another severe attack of melancholia, which interrupted the work for a year.[7] He again tried to hang himself, but was cut down by Mrs. Unwin.[8]

The successful translation of Homer into English verse has been deemed by good scholars to be impossible. Goldwin Smith said that the attempt has always failed. There is something in the Greek epic—its remote age, primitive culture, barbarous fighting, preposterous gods, and especially the barrier of language and verse—that has made its translation an abortive scheme. Pope had translated Homer into jingling English rhymes, but his work is no more like the Greek epic than a doll's house is like a Greek temple. Bentley, the great classic scholar, said of it, "It is a very pretty poem, Mr. Pope, but you must not call it Homer." Cowper, knowing all this, would improve on Pope and chose English blank verse as his medium. Goldwin Smith thinks that it was under an evil star that he took to this work, but Cowper himself said that he began it as a diversion. He worked at it for many hours every day, and until the very end of his life devoted himself to its revision. His letters during this time were often despairing even to incoherence in his religious gloom, and fill the mind with amazement that a man in such a state could apply himself to any intellectual occupation.[9] It is the best evidence one could have that Cowper had suffered no mental deterioration. His translation has been praised for its accuracy, even though it is lacking in the true Homeric ring. Almost the last glimpse we have of him is when toward the very end

6. Rush, B.: Diseases of the Mind, ed. 4, Philadelphia, J. Grigg, 1830, p. 119.

7. Southey (footnote 3, vol. 2, p. 129).

8. Stephen, Leslie: Cowper, in Dictionary of National Biography.

9. Cheever, G. B.: Life, Genius and Insanity of Cowper, New York, R. Carter & Bros., 1856, pp. 384, 385.

of his life he was still revising his work.[10] It is said that the last line he ever wrote was in the last year of his life, when he revised his translation of a passage in the "Iliad." He was eventually removed from Olney to Weston, and his life during his later years was largely planned for him and controlled by others. He was one of the best of letter writers and left not a few letters which reveal his mental state.

A curious episode in Cowper's career was his affair with a man named Teedon. This man had been a schoolmaster at Olney, and is described as a dull, conceited fellow, whom Cowper had long known, endured and even laughed at. But after the poet, at 60 years of age, had published his Homer, and was falling more and more into a state of chronic despair, Teedon induced him to believe that he was receiving communications from heaven in Cowper's behalf. These oracles were duly transmitted to Cowper, who so firmly believed them to be genuine that he copied them into a book. Mrs. Unwin also came to share in this belief; and the three of them formed a coterie, communicating their beliefs one to another, an instance of what may be called mental contagion. But all these mysteries did no permanent good. Cowper's chief delusions were unchanged, and were accompanied with alarming auditory hallucinations, all of which persisted until his death.[11]

He came in time to believe that he was doomed not to die but to be carried off alive and cast bodily into perdition. He sometimes sat mute for days, refusing food.

What shall one call the case of Cowper? It can hardly be called a case of manic-depressive insanity. The evidence of an acute manic phase is lacking, yet there was sometimes a mild hypomania following his melancholic attacks. All through his life he seems to have had periods of high spirits, when his religious fears and horrors were temporarily repressed. But the gloomy, repressed emotions were never entirely absent, although their activity varied. They were always just beneath the surface, not exactly subconscious, but enough under control at times to permit a normal action of mind. This is a freudian aspect of his case, which is noteworthy. They influenced Cowper's life for more than thirty years. He could never free himself from their tyranny, and he always misinterpreted them as genuine religious experiences. Thus they became fixed delusions. For long years there was no evidence of dementia. He could concentrate his mind on his Homer and work at it every day for five hours at a stretch, conscious all the while of the

10. Spiller, R. E.: A New Biographical Source for William Cowper, Publication of the Modern Language Association of America, December, 1927.

11. Southey (footnote 3, vol. 2, p. 213). Here are found many of the poet's morbid letters to his cousin, Lady Hesketh, and others. H. Crabb Robinson's Diary, Boston, Osgood & Company, 1871, vol. 2, p. 67.

haunting fears that obsessed him.[12] The case is probably best described as a form of circular insanity, with alternating phases of profound depression and mild hypomanic reaction, but without any distinct intervals of complete sanity, because these delusions were always lurking in his mind. It was a constitutional psychosis, call it what one may.

It was not until toward the end of his long life of nearly 70 years that Cowper's mind finally gave way in dementia; and this final stage presents the appearance of an involutional melancholia.[12a]

12. Southey (footnote 3, vol. 2, p. 77). Here appears one of his despairing letters written at the time he was translating Homer.

12a. Haley's "Life and Letters of Cowper" is the best original source, and gives the dark picture of the poet's declining years (vol. 4, p. 138, etc.). See also Cecil, D.: The Stricken Deer, Indianapolis, Bobbs-Merrill Company, 1930; Bradford, Gamaliel: Cowper, in Bare Souls, New York, Harper & Brothers, 1924, p. 133.

STAINING NERVE FIBERS IN MOUNTED SECTIONS WITH ALCOHOLIC SILVER NITRATE SOLUTION*

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The procedures to be described have developed through experiments designed to test the effects of varying some of the many conditions involved in silver staining. The previous article [1] on staining nerve fibers in unmounted celloidin sections gave the technic from which the present method has evolved.

The application of silver solutions to mounted sections with subsequent reduction of adsorbed or chemically combined silver salt is not new. Sand [2] embedded nerve tissue in paraffin after fixation in a strong solution of nitric acid in acetone and later impregnated the mounted sections. Malone [3] described a method for handling paraffin sections to produce a Cajal type of stain. Brandt and Kadanoff [4] investigated the effect of varying the time of impregnation and the concentration of the silver nitrate bath on sections from celloidin, paraffin and gelatin embedded material. They chose gelatin as best suited to the O. Schultze method. More recently, Foot [5] described a method for staining by silver tannate paraffin sections from Zenker-fixed material of the central nervous system.

Two other methods for staining nerve fibers in mounted sections have come to my attention since submitting this article: Freeman's [6] method and Kernohan's [7] adaptation of Bielschowsky's method to embedded material.

The method to be described differs from preceding methods in the fact that alcohol is used as the chief solvent for the silver nitrate and the reducing agents. The chief advantage of this is that it allows the reduction of the silver nitrate to be more easily controlled than in aqueous solutions, because the ionization of the reactants is much less in alcohol than in water. Another purely technical advantage lies in the elimination

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* From the Institute of Neurology, Northwestern University Medical School.

1. Davenport, H. A.: Anat. Record **44**:79, 1929.
2. Sand, R.: Arb. a. d. neurol. Inst. a. d. Wien. Univ. **14**:339, 1908.
3. Malone, E. F.: Anat. Record **9**:791, 1915
4. Brandt, W., and Kadanoff, D.: Ztschr. f. Anat. u. Entwickl. **66**:211, 1922.
5. Foot, N. C.: Silver Tannate Technic for Paraffin Sections from Central Nervous System, Arch. Path. **4**:36 (July) 1927.
6. Freeman, W.: Silver Diffusion Method for Staining Nerve Fibers in Paraffin Sections, Arch. Neurol. & Psychiat. **7**:321 (March) 1922.
7. Kernohan: Tr. Am. Micr. Soc. **49**:58, 1930.

of the necessity of running the material down to water and back again. The use of alcohol in the silver bath seems to have been originated by Cajal,[8] although the solution described by him contained only about 30 per cent alcohol, while I have used solutions which had an alcohol concentration of from 85 to 90 per cent.

The use of alcoholic silver nitrate, particularly when the solution is slightly acidified with nitric acid, accentuates the staining of nerve fibers and nuclei with relative suppression of other tissue elements. The technic is suited for the demonstration of nerve fibers in either paraffin or celloidin embedded material. Formaldehyde-fixed material has been used for working out the method in order that it might fit the needs of the neuropathologist.

The following points seem to deserve emphasis: (1) the provision of a diffusion zone of uniform thickness between the section and the staining reagents, (2) adaptation of the proper acidity of the silver bath to the particular tissue to be stained, (3) time in the silver bath, (4) degree of reduction and (5) prevention of surface precipitate. These features will be taken up in detail under "procedures." An attempt has been made to simplify the technic as much as possible and to eliminate unnecessary reagents. However, the simplicity has not been carried to a degree incompatible with obtaining good results on a rather wide range of material.

PROCEDURE

(1) The preparation of the sections for silver impregnation consists of mounting them on slides and coating them with celloidin. If celloidin sections are used, they can be mounted with albumin fixative as in the method used by Maximow,[9] but instead of covering the sections with oil, the slides are plunged immediately into a solution of 2 per cent celloidin. In mounting, care should be taken not to wash off too much of the albumin-glycerin fixative with the 70 or 80 per cent alcohol from which the sections are taken. By covering the middle of the slide with fixative, immersing one end of it in the dish containing the sections and drawing a section up over the portion covered with fixative, a minimum amount of alcohol is carried along and good fixation secured. The section is pressed with bibulous or other lintless blotting paper from the center outward to avoid wrinkles. All excess alcohol should be removed, and before the edges curl, the slide is put into the dilute celloidin. When the edges of the original celloidin soften, the slide is removed, drained quickly, inverted, and tilted back and forth so that the coating will flow evenly, and then laid face up (supported at the ends) until the coating sets. When well set and before the drying of the surface begins, the preparation is put into a solution of 80 per cent alcohol. After a few minutes, it is ready for the silver bath.

The mounted paraffin sections are passed through xylene, absolute alcohol, alcohol-ether and 2 per cent celloidin. They are manipulated as are the celloidin sections to get the coating as even as possible.

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8. Cajal, S. Ramon: Ztschr. f. d. ges. Neurol. u. Psychiat. **100**:738, 1926.
9. Maximow, A.: Ztschr. f. wissensch. Mikr. **26**:177, 1909.

The celloidin coating provides a diffusion zone between the material being stained and the reducing solution. It is also a removable covering which keeps the precipitate from the surface of the section during the development, and its subsequent removal as a rule eliminates all the fine surface precipitate often seen in frozen sections and unmounted celloidin sections.

(2) The silver bath consists of a solution of 10 per cent silver nitrate in 85 per cent ethyl alcohol slightly acidified with nitric acid. Dissolve 10 Gm. of (reagent grade) silver nitrate in 10 cc. of distilled water and add 90 cc. of 95 per cent alcohol. A solution of the salt is more easily effected in the small amount of water, because it is much more soluble in water than in alcohol. The strength of the bath is greater than necessary, but the solution can be used repeatedly, and some silver nitrate will be carried out by the tissue. A strong solution has been found more satisfactory than a weak one, because it shortens the time necessary for impregnation. The acidification of the silver bath permits greater differentiation between nerve fibers and neuroglia. Prepare an approximately normal (N/1) (7 per cent concentrated) aqueous solution of nitric acid and, if staining is done in Coplin jars (50 cc.), add from 5 to 7 drops of the acid solution to each jar. Since the size of drops varies, a more accurate way is to add 0.5 cc. of acid to each 100 cc. of alcoholic silver solution. This amount has been found suitable for most tissue, although some has required only half as much or even none, while one specimen of human cerebellum required about 0.8 cc. of acid per hundred cubic centimeters of silver bath for best results. When too much acid has been added, the nuclei develop too dark and the stain may appear patchy. Too little acid is characterized by lack of contrast between nerve fibers and other structures. It is not known whether the acidification of the silver bath affects the adsorption of the silver salt by the tissue or more indirectly affects the rate of reduction in the developing solution, but the former seems the more probable. When acid is added directly to the silver bath, no pretreatment with strong nitric acid (as previously recommended [1]) is necessary.

(3) The time the material is to be left in the bath can be judged by the appearance of a light brown tint in the tissue. At temperatures of from 37 to 40 C., formaldehyde-fixed material may require only one hour. One specimen of alcohol-fixed material required four days. The shortest possible time compatible with good staining of nerve fibers is to be preferred to overimpregnation, for the latter may lead to poor differentiation. The apparent difficulty of the uncertainty of time in the silver bath can be overcome by preparing three extra slides and developing one in one hour, one in four hours and one in an overnight period. The first to show sufficient silvering indicates that the rest should be developed at that time. A little experience in judging the appearance of the brown tint in the tissue will prove to be a fairly dependable guide as to the correct impregnation time.

By most methods, the impregnation is carried out in the dark. It seems that the effect of light has been somewhat overemphasized, although the avoidance of direct sunlight or long exposure to direct daylight would seem good practice in this procedure. The incubator used in this laboratory has been dark, but no precautions have been taken relative to exposure to diffuse daylight during development, and no effects attributable to light have been observed.

(4) After removing from the silver bath, rinse quickly in strong alcohol (95 per cent to absolute) and reduce in an alcoholic solution of a mixture of formaldehyde and pyrogallic acid. A solution consisting of 5 Gm. of pyrogallic acid, 5 cc. of neutral formaldehyde solution and 100 cc. of 95 per cent alcohol has been satisfactory. Various combinations of hydroquinone and formaldehyde and

formaldehyde alone have been less satisfactory, as they worked too slowly and often gave a coarse grained reduction product.

The time of reduction, like the development of a photographic image, is one of the important phases of section staining. The nerve fibers appear first, and reduction should not be allowed to progress until neuroglia tissue becomes dark. The degree of development can best be controlled by observation under the microscope, but it can be judged macroscopically by the intensity of the brown color. Some tissue, especially when the time and acidity of impregnation have been optimal, have little tendency to overdevelop. Other specimens may require only a minute or two in the reducing solution, and since the reduction continues slowly for several minutes after the specimen is transferred to 95 per cent alcohol, it should be removed from the reducing solution somewhat underdeveloped when it turns brown within the first minute or two. Diluting the reducing solution with

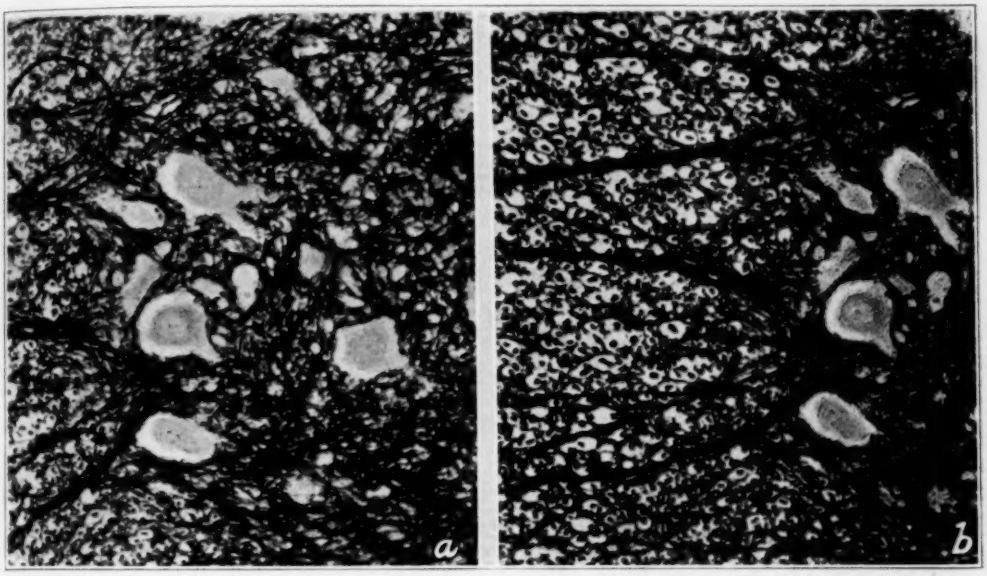

Fig. 1.—*a*, low power photomicrograph of the cross-section of the spinal cord of a cat at the margin of the anterior gray column. The fiber is stained by alcohol-silver process in paraffin section. Photographed with Leitz objective × 10, ocular × 8, filter VL. *b*, a similar field without filter.

an equal volume of alcohol provides a means of controlling too rapid development. Longer impregnation in the silver bath is indicated when the sections require more than ten minutes' reduction to bring out the nerve fibers.

(5) The formation of precipitates on the surface of sections is frequently a source of despair. This trouble has been reduced to a minimum by the addition of a colloid to the reducing solution, as recommended by Liesegang[10] for frozen sections, and by the celloidin coating and its subsequent removal. The precipitate which remains on the surface in spite of the colloid is removed if the slide is kept in motion while the celloidin is being dissolved. Keeping the slides in motion during development is helpful also.

10. Liesegang, R. E.: Kolloïdschem. Beihefte **3**:1, 1911.

The colloid used has been dextrin and the more hydrolyzed type such as found in corn syrup has been more satisfactory than the chemically pure grade. The latter gives a coarse precipitate in the alcoholic reducing solution and aggregates too rapidly. The corn syrup is diluted with two or three parts of water, and a few drops of this solution is added to the reducing solution from time to time. The colloidal dextrin which forms in the reducing solution appears to bear an

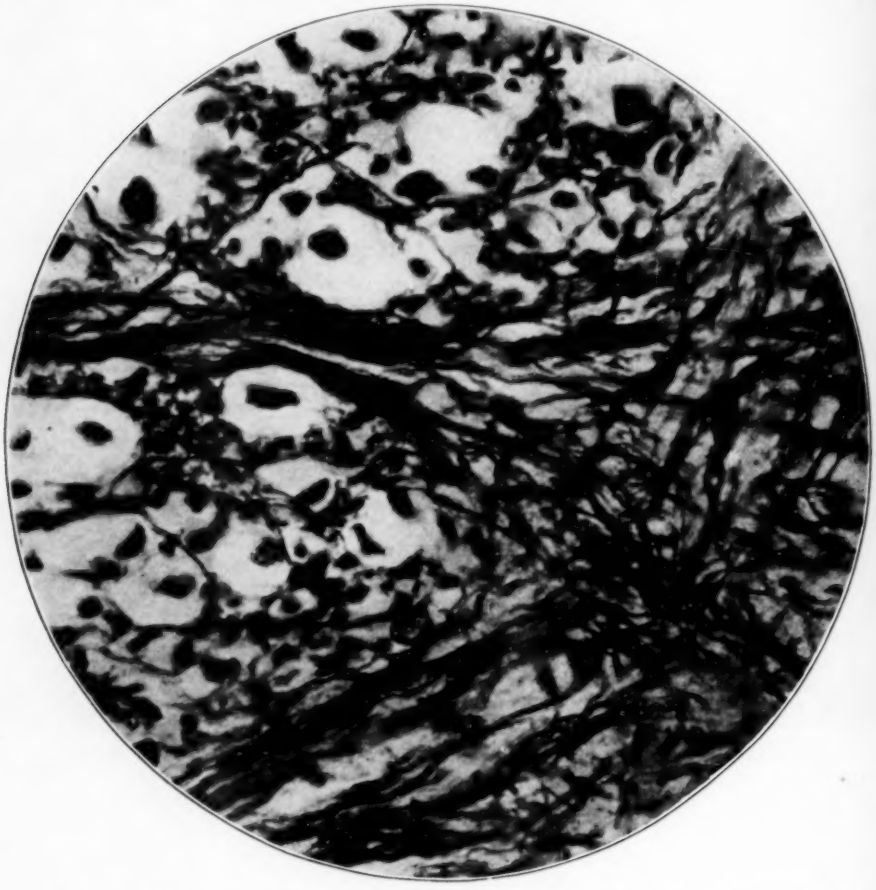

Fig. 2.—High power photograph of enclosed area in figure 1. Photographed with yellow filter; Leitz 2 mm. objective, ocular ×8. Filter VL.

electrical charge opposite that of the precipitated silver, hence forms aggregates with it and thereby prevents it from being deposited on the surface of the slide. If the reducing solution is kept turbid with dextrin, at least twenty specimens can be developed in 50 cc. without renewal.

After development the preparations are passed through two or three changes of 95 per cent alcohol, giving two or three minutes to each in order to remove the reducing agents, then into absolute alcohol. The celloidin coating is removed with a mixture of equal parts of absolute alcohol and anhydrous ether. The slides

should be kept in motion if practicable during immersion in the first jar of alcohol-ether and then rinsed clean in a second solution. Xylene and balsam follow. No treatment with "hypo" is necessary in the process described, although some clearing of the background can be effected with acid "hypo" made up according to any photographic formula. Acid "hypo" which contained alum has effected the greatest improvement in differentiating if before being used it was allowed to evaporate in a shallow dish until a precipitate began to settle out. Probably the simplest way to accentuate contrast in the preparations is by gold toning. Reddish to violet tones can be obtained by using a dilute acidified gold solution, while grays and black prevail when the more concentrated or alkalinized gold solutions are used.[11]

Comment.—Although the method described was developed chiefly on material taken from the central nervous system of man and cat, it has given similar results on the spinal cord of a dog and the brains of the guinea-pig and rabbit. It has been used successfully to demonstrate nerves and motor end-plates in the voluntary muscle of the cat, but has not yet been successfully adapted to peripheral nerves in other tissues. The best results on muscle were obtained when the blood had been washed out by perfusion with saline solution, fixed for one week in 10 per cent of solution of formaldehyde and embedded in paraffin.

The chief difficulties found with the method were that it seldom demonstrated neurofibrils and synaptic end-loops, and sometimes failed to stain the very finest fibers of gray matter.

The advantages were the adaptability to autopsy material without the necessity of providing special blocks or frozen sections for nerve fiber staining, and to serial section work when the alternation of a silver stain with other stains is desired.

The illustrations indicate the character of differentiation. The use of a yellow filter in photographing has suppressed the cells (stained yellow) and accentuated the fibers (stained dark brown to black).

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11. Bielschowski, M., and Pollack, B.: Neurol. Centralbl. **23**:387, 1904.

EXPERIMENTAL LESIONS IN THE TUBER CINEREUM OF THE DOG

FOLLOWED BY EPILEPTIFORM CONVULSIONS AND CHANGES IN BLOOD CHEMISTRY *

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Certain animals operated on by one of us,[1] in a previous investigation concerned with the nuclei of the subthalamus, exhibited periodic convulsions following the operation. It was noted that in those animals there was always injury to the region of the tuber cinereum. Following unilateral lesions in the tuber cinereum, the symptoms were so generalized and complex and the various symptoms combined in such a uniform manner that it seemed impossible to explain the convulsion on the basis of specific nuclei and fiber connection for each symptom (a center for pupillary dilation, one for salivation, another for vasoconstriction, another mechanism for the muscular spasm, etc.). On the contrary, it seemed more likely that there was some more generalized mechanism in the tuber cinereum which exerted an influence on the body by means of substances carried in the blood stream. It seemed possible that this might be in the form of an excessive amount of urea or other toxic substance in the blood, a disturbance of the normal potassium or calcium content of the blood, or perhaps a disturbance in the normal function of the endocrine glands.

This investigation was undertaken with the purpose of studying the blood chemistry following lesions in the tuber cinereum, determining, if possible, what specific cell groups in the tuber cinereum are responsible for the symptomatology, and defining, as far as possible, the mechanism by which the changes in blood chemistry and convulsions are produced. Incidentally, it is believed that we have obtained some indications as to the normal function of the cell groups in the tuber cinereum.

Many workers have produced experimental convulsions by the use of drugs, such as absinth, wormwood oil, picrotoxin, acid fuchsin and

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1. Morgan, L. O.: Symptoms and Fiber Degeneration Following Experimental Lesions in the Subthalamic Nucleus of Luys in the Dog, J. Comp. Neurol. **44:** 379, 1927.

strychnine, by ligating the carotid arteries and by stimulating the cerebral cortex. Since we are unable at present to say what relationship, if any, exists between the convulsions produced by those methods and the convulsions that follow lesions in the tuber cinereum, we shall not attempt to discuss the literature on that subject at the present time. Muskens [2] and Lennox and Cobb [3] have presented excellent reviews of present knowledge concerning the nature of convulsions from the standpoint of both experimental convulsions and the convulsions occurring in epilepsy in man.

The convulsions occurring after lesion in the tuber cinereum in our experimental animals and their similarity to epileptic convulsions in the human being suggested the possibility that the tuber cinereum might in some way be concerned with idiopathic epilepsy in man. Morgan [4] described a marked pathologic change in the brain in six cases of idiopathic epilepsy and discussed the possible relationship of the tuber cinereum to epilepsy. In this paper an attempt was made to review some of the most important literature touching on the function of the tuber cinereum. Later, Morgan and Gregory [5] reported pathologic observations in the tuber cinereum in twenty-one additional cases of idiopathic epilepsy.

MATERIAL AND METHODS

A successful lesion was placed in the tuber cinereum of sixteen dogs. Twelve of these cases are described in this paper. The lesions were made by the method previously described by one of us.[1] Three different approaches were used in reaching the tuber cinereum.

The method most frequently used was to reflect the dorsal part of the temporalis muscle, trephine a small opening over the parietal region of the brain and project a hypodermic needle from this point through the cerebral cortex and along the dorsal side of the internal capsule into the region of the tuber cinereum. The needle, in most cases, was run through the dura without making an incision in this membrane. External landmarks on the skull were used in calculating the proper angle at which to insert the needle. A second method of approach was to insert a needle through the orbit, thence back through the optic foramen to the base of the tuber. The third approach was through the lower temporal region of

2. Muskens, L. J. J.: Epilepsie; vergleichende Pathogenese, Symptome, Behandlung, Berlin, Julius Springer, 1926; English edition, New York, William Wood & Company, 1928.

3. Lennox, W. G., and Cobb, Stanley: Epilepsy; From the Standpoint of Physiology and Treatment, Medicine **7**:105, 1928.

4. Morgan, L. O.: The Nuclei of the Region of the Tuber Cinereum: Degenerative Changes in Cases of Epilepsy, with a Discussion of Their Significance, Arch. Neurol. & Psychiat. **24**:267 (Aug.) 1930.

5. Morgan, L. O., and Gregory, H. S.: Pathological Changes in the Region of the Tuber Cinereum in Idiopathic Epilepsy, Am. J. Psychiat **9**:805 (March) 1930.

the skull as described by Crowe, Cushing and Homans [6] for hypophysectomy. The brain was raised from one side and the tuber cinereum reached from its base. In any case 0.1 cc. of from a 0.2 to 0.3 per cent solution of mercuric chloride was injected as the means of producing the lesion.

Since the convulsions usually began soon after the operation, it was found that in using the lower temporal approach the temporary injury to the brain incident to the operation made it difficult to determine what part of the symptomatology was due to the lesion in the tuber cinereum. In employing the orbital and parietal approach the difficulty of accurately localizing the lesion in the tuber cinereum was so great that only about one third of the animals operated on proved to have a successful lesion in this region. As a result we have about forty dogs which showed a great variety of lesions in the regions adjacent to the tuber cinereum. The symptomatology and lesion in these animals were compared with those of the animals with lesions in the tuber cinereum and were useful in isolating symptoms due to injury in the tuber cinereum from symptoms due to injury in adjacent areas. These animals were also used as controls in studying the blood chemistry.

After the animal died or was killed, the brain was fixed in 10 per cent of a commercial formaldehyde solution, embedded in celloidin and sectioned for microscopic study. The sections were stained by the iron-hematoxylin method previously described by Morgan,[4] giving both a nerve cell and myelin sheath stain.

NORMAL ANATOMY

Figure 1 is a photograph of a sagittal section through the head of a dog showing the position of the tuber cinereum in relation to the base of the skull, as well as to adjacent parts of the brain. Figure 2 is a stained cross-section through the diencephalon striking the tuber cinereum about midway between the optic chiasm and the mammillary bodies.

The region with which we are dealing reaches from the optic chiasm caudad, past the anterior half of the mammillary bodies. It extends from the base of the brain to the ventral side of the thalamus. It is bounded medially by the third ventricle and laterally by the medial side of the internal capsule and the medial side of the subthalamus composed largely of Forel's fields H^1 and H^2 and the zona incerta. The fornix passes through this area to reach the mammillary body.

The cell groups in this region in the dog more closely resemble those in the human brain than in any other form that has been described.

6. Crowe; Cushing and Homan: Experimental Hypophysectomy, Bull. Johns Hopkins Hosp. **21**:127, 1910.

Malone [7] described three cell groups in the region of the tuber cinereum in man: the substantia grisea of the third ventricle, the nucleus tuberis lateralis and the nucleus tuberomammillaris. Similar cell groups were later described by Greving [8] and others. The cell groups in the region of the tuber cinereum in the dog differ in some respects from those in man, but it is believed that they correspond to the three nuclei described by Malone and Greving.

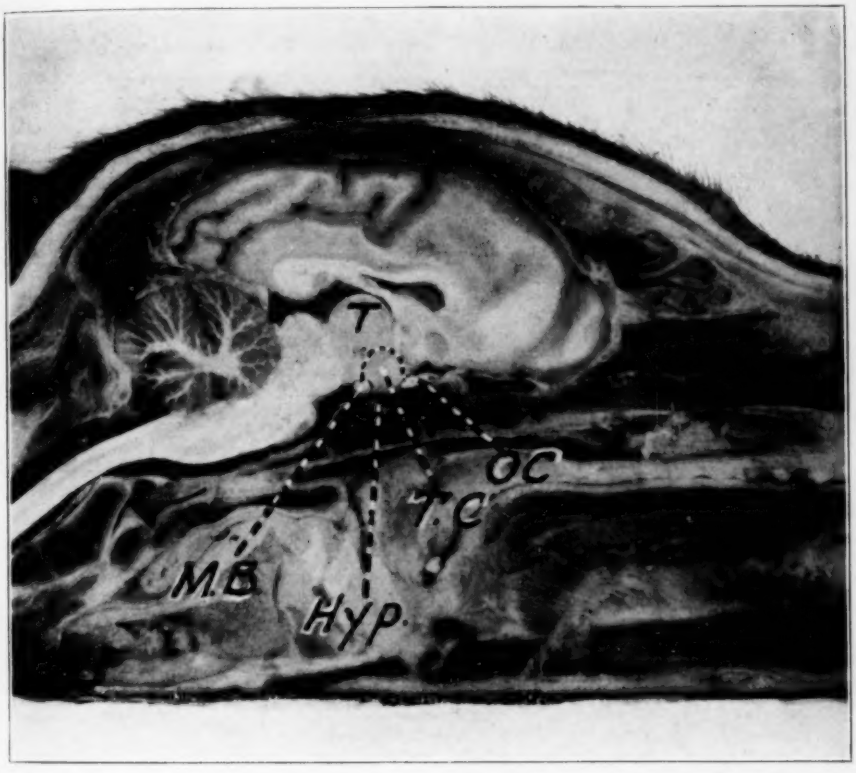

Fig. 1.—Sagittal section through the head of a dog, showing the relations of the tuber cinereum to the base of the skull and to adjacent parts of the brain. *O.C.* indicates optic chiasm; *T.C.*, tuber cinereum; *Hyp.*, hypophysis; *M.B.*, mammillary body; *T.*, thalamus.

The substantia grisea is composed of small rounded or oval cells with comparatively large nuclei and scant cytoplasm. The cytoplasm

7. Malone, E. F.: Ueber die Kerne des menschlichen Diencephalon, Abhandl. der königl. Preuss. Akad. d. Wissensch., suppl., 1910; The Nuclei Tuberis Laterales and the So-Called Ganglion Opticum Basale, Johns Hopkins Hosp. Rep., Monograph No. 6, 1914.

8. Greving, R.: Beiträge zur Anatomie des Zwischenhirns und seiner Funktion, Ztschr. f. d. ges. Neurol. u. Psychiat. **99**:231, 1925.

contains small Nissl bodies which do not stain darkly or stand out prominently. These cells extend from the base of the brain to the thalamus and from the optic chiasm to the mammillary bodies. This nucleus is medially located in the immediate wall of the third ventricle. There is a tendency for the cells to be more concentrated in certain areas. One such area is located in the basilar part of the wall of the ventricle, a second in the region ventromedial to the fornix, while a third and smaller group is located medial to the fornix. Laterally, the cells of this nucleus thin out and become scattered among the cells of the other nuclei.

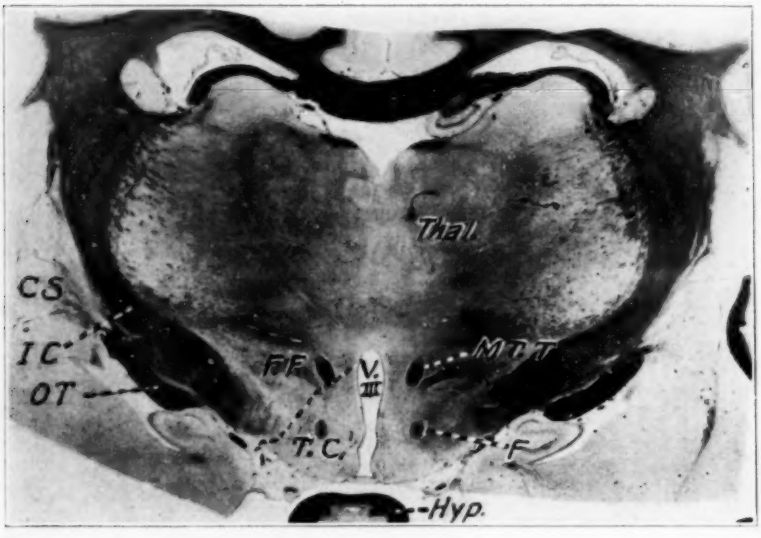

Fig. 2.—Cross-section through the diencephalon of the brain of the dog stained with iron hematoxylin. *C.S.* indicates corpus striatum; *I.C.*, internal capsule; *O.T.*, optic tract; *F.F.*, Forel's field; *T.C.*, tuber cinereum; *V.III.*, third ventricle; *Thal.*, thalamus; *M.T.T.*, mammillothalamic tract; *F.*, fornix; *Hyp.*, hypophysis.

The nucleus tuberis lateralis is composed of cells which are larger than those of the substantia grisea and contain a relatively small nucleus and large amount of cytoplasm. The cells are more elongated in form. The chromatin has a tendency to accumulate in elongated masses, which are usually parallel with the long axis of the cell. These masses of chromatin frequently stain very dark. In the human being the nucleus tuberis lateralis forms a number of well circumscribed cell groups which lie along the lateral basilar part of the tuber cinereum. While in the dog there is a tendency for the cells to be more concentrated along the lateral basilar portion of the tuber cinereum, there are no well

defined circumscribed cell groups, and the cells are scattered dorsally through a large area surrounding the fornix.

The cells of the nucleus tuberomammillaris are much larger than those of either of the other nuclei described. These cells are usually somewhat oval in form and contain a large nucleus and a large amount of cytoplasm. The cytoplasm is filled with large Nissl bodies which take a deep stain and are irregular in shape. There is a tendency for these cells to concentrate around the fornix, and smaller groups of cells are found along the base of the tuber cinereum and in the region dorsal to the fornix. At the anterior end of the mammillary body, the cells of the nucleus tuberomammillaris collect along the ventrolateral side of that structure and extend past the anterior half of the mammillary body.

PROTOCOLS OF EXPERIMENTS

Dog 3 A.—October 10: At 11 a. m., 0.1 cc. of a 0.3 per cent solution of mercuric chloride was injected into the region of the tuber cinereum through the left parietal lobe.

Symptoms.—Beginning at 2:30 p. m., the animal had five mild convulsions at intervals of about five minutes. Each of these lasted about thirty seconds or more and involved only the fore part of the body. The front legs would be extended rigidly, the head thrown back, the jaws would snap open and shut, the muscles of the face and neck would twitch and there was frothing at the mouth. The left pupil was dilated, but the right was contracted. The muscles on the right side of the body were hypertonic.

Following these light attacks, there occurred a severe seizure lasting for more than one minute. The muscles suddenly became rigid, and the animal fell on the side, frothing at the mouth. There was vasoconstriction, and the penis was erected. The animal was unconscious and required a few minutes after the convulsion to regain consciousness. The temperature at this time was 103.3 F., and the pulse rate 120. During the next hour, there was continued frothing at the mouth, vasoconstriction and erection of the penis, and the animal was somewhat disoriented. A large quantity of watery feces was passed. Several light convulsions occurred during this time. The following morning the dog was found dead in the cage.

Lesion.—Only a macroscopic study of the brain was made in this case. There was a fairly large softened area lateral to the anterior end of the mammillary body. The lesion would encroach on the caudal part of the tuber cinereum. The caudolateral extension of the lesion probably involved the medial region of the subthalamic nucleus of Luys.

Dog 8 A.—October 17: At 10 a. m., 0.1 per cent solution of mercuric chloride was injected through the left orbit in an attempt to reach the tuber cinereum.

Symptoms.—At 2:30 p. m., in walking, the left hind leg was stiff and moved from the hip. The left front leg was weak under weight and inaccurate in movement. The right hind leg was slightly spastic. The heart rate was 122; the temperature, 101.9 F. The right pupil was completely dilated. The skin and oral epithelium was blanched, indicating vasoconstriction.

At 8:50 p. m., there was excessive salivation and frothing at the mouth. The animal had a wild, frightened appearance. The dog was having mild spasms at long irregular intervals. These involved the fore part of the body only.

At 10 p. m., the animal was having fairly severe seizures at intervals of about thirty minutes. These involved the entire body but did not cause the animal to fall. The dog became confused and disoriented but did not completely lose consciousness. The attack began by the animal standing with the muscles rigid. This was followed by clonic spasms which caused the animal to lose its balance but not to fall. These seizures were accompanied by frothing at the mouth, vasoconstriction, dilatation of the right pupil and erection of the penis. The seizures left the animal hypertonic with tremor over the entire body.

October 18: At 1:20 p. m., the animal was observed to be panting rapidly (240 respirations per minute). The tongue was flushed. The animal was extremely restless, and there was a coarse tremor over the entire body which ceased with activity. The muscles of the entire body were tense and hypertonic. The animal was hypersensitive to touch and pressure. The heart rate was 215 and the temperature 105 F.

At 8:20 p. m., the heart rate was 132, respiration 54 and temperature 105.3 F. The animal was somewhat disoriented and restless. The right pupil was dilated, and the left was almost completely dilated. Hypertonicity of the muscles was evident only during activity or when the animal was stimulated by jerking the foot or stimulating the skin. The skin and oral epithelium seemed to be flushed.

October 19: At 3 p. m., the heart rate was 162, the respiration 9 and the temperature 100.8 F. The right pupil was completely dilated; the left was half dilated. The animal lay on the side in a state of coma. It did not react to painful stimulation but gave the leg retraction reflex when the foot was jerked. The head was thrown back and to the left with a rigidity of the muscles on that side of the neck. When the abdominal wall was opened, the intestines and stomach were observed to be relaxed and flaccid. The animal was killed.

Lesion.—The needle passed into the inferior horn of the lateral ventricle of the left side. The mercuric chloride was injected entirely into the lateral ventricle. There was much injury and destruction throughout the walls of the left ventricle. In addition to this, sufficient fluid reached the third ventricle to cause marked injury also in its walls. A narrow zone extended along the third ventricle on each side in which there was marked vascular congestion and almost complete destruction of the nerve cells. There was considerable increase in the number of glia cells, and the connective tissue element seemed to suffer little. In this zone bordering the third ventricle, however, there were no nerve cells, except a few abnormal ones, and many nuclear remnants. In a second zone, lateral to the medial one, there were many cells showing a varying degree of injury with little total destruction. The substantia grisea would suffer most in this case, although there would be injury to the more medial part of the other two cell groups (nucleus tuberis lateralis and nucleus tuberomammillaris).

Dog 9 A.—On October 24, at 9:30 a. m., 0.1 cc. of a 0.3 per cent solution of mercuric chloride was injected into the region of the tuber cinereum through the left orbit.

Symptoms.—On regaining consciousness following the operation, the animal sat rather rigidly on its haunches with the head drawn back, and there was a constant driveling of saliva.

At 1:15 p. m., the animal appeared to be normal in its reactions, except that a coarse tremor occasionally passed over the body and the left pupil was dilated (perhaps caused by injury to the optic nerve). The heart rate was 120, the temperature 102 F. The animal had three moderately severe convulsions during the next twenty minutes. These involved primarily the fore part of the body.

Salivation and vasoconstriction were prominent. The dog became disoriented but did not lose consciousness. These attacks were followed and sometimes preceded by a coarse tremor.

At 1:35 p. m., there was a prolonged severe seizure lasting for about four minutes. This was followed, two minutes later, by a second attack which lasted about half as long as the first. These were characterized by unconsciousness, frothing at the mouth, dilatation of the pupils, vasoconstriction and increased heart rate. Since the operation, defecation had occurred four times, and there was frequent urination.

Convulsions continued to occur at brief intervals. The animal remained in a dazed condition between attacks. During these intervals, there was more or less intermittent twitching of the muscles of the body. This was much more pronounced in the fore part of the body. Frequently the muscular twitching would begin on the face, increase in severity and pass over the remainder of the body. The muscles would then pass into rigidity, and a convulsion would follow. The heart rate was about 180 during the intervals and would rise to 240 or more during the convulsion. The temperature was 104.7 F.

At 5 p. m., the animal was in coma and convulsions were occurring more frequently. The heart rate was about 140 in the intervals between attacks and 205 or more at the end of a convulsion. The temperature was 103.2 F. Excessive salivation, dilatation of pupils and vasoconstriction were constant.

At 6 p. m., the animal was in an almost continuous clonic spasm. The muscular twitchings were occurring at the rate of about 168 per minute. The temperature was 108.5 F. The abdominal wall was opened in order to examine the viscera. The intestines and stomach were distended and flaccid. The bladder was contracted, but the wall showed only a low grade of tonicity. At 6:30 p. m., the muscles were in one continuous clonic spasm. The heart rate was 312, and the temperature ran beyond what the thermometer could register (111 F.). The animal died a few minutes later.

Lesion.—The needle passed through the optic foramen into the plexus of blood vessels at the base of the tuber cinereum. The mercuric chloride was injected against the basilar surface of the tuber cinereum and spread along the surface on both sides. A mass of brown coagulated substance filled the subarachnoid space along the base of the tuber cinereum on each side and continued caudal to the mammillary bodies as far as the beginning of the pons. No gross lesion was present, but a great deal of cell destruction and injury was evident in a zone extending along the basilar surface of the tuber cinereum on each side. In many instances cell loss was complete, only the cell space or a small remnant of the cell remaining to indicate where the cell had been. Many cells showed degenerative changes of varying degrees. The nuclei were usually shrunken, darkly staining and lay at the periphery of the cell. In some cases the cytoplasm showed the characteristic changes of chromatolysis. In other cells there was a swollen appearance with the cytoplasm precipitated in granular threads. The zone affected contained many cells of each the substantia grisea, nucleus tuberis lateralis and nucleus tuberomammillaris.

Dog 19 A.—November 28: At 9:45 a. m., 0.1 cc. of a 0.3 per cent mercuric chloride solution was injected into the region of the tuber cinereum by way of the left parietal region.

Symptoms.—At 1:20 p. m., the animal stood in a tense attitude, with the head being jerked about in a quick nervous manner suggesting fear and disorientation. There was excess salivation, and the pupils were dilated. Mild convulsions involving the fore part of the body were occurring at intervals of from six to eight

minutes. When released from the cage, the animal ran about frantically as if in fear.

At 2 p. m., the rate of heart beat was 168 and irregular. The temperature was 106.6 F. The animal was panting rapidly, was restless and appeared to be confused and frightened. There was a tendency for the body to bend to the right and for the animal to turn to the right. Convulsions were occurring at intervals of from three to four minutes. These were more severe, frequently involving the entire body. Although the animal lost apparently all grasp of its surroundings and staggered about during the attack, it did not completely lose consciousness or fall. In the intervals between seizures, the head was moved about constantly in a quick, jerky manner, and the animal was constantly shifting about in a restless, hypertonic manner. There was a frequent twitching of various muscles and groups of muscles. There was constant excessive salivation.

At 5 p. m., the symptoms had gradually diminished in severity. The dog had apparently almost regained the grasp of its surroundings. The muscles were much more relaxed although still somewhat hypertonic. The heart rate was 150 and the temperature 104.8 F.

At 9 p. m., the temperature was 104.6 F. and the heart rate 120. Two mild convulsions occurred at fifteen minute intervals.

November 30: The muscles were stiff in movement, and the animal walked with the nose thrust out and the hips drawn up. The hind feet were dragged along close to the floor in a shuffling manner, and the animal staggered when it walked. At times there was a coarse tremor over the entire body. The pupils were nearly normal. The heart rate was 144 and the temperature 101.6 F. At 1:45 p. m. the dog had a severe seizure which lasted for about four minutes. The animal fell on its side unconscious, passing through both the tonic and the clonic phases of the seizure. The usual symptoms such as excessive salivation, dilatation of the pupils, vasoconstriction and increase in heart rate were present. The seizure was followed by a coarse tremor which lasted for about an hour.

December 1: The animal showed a coarse tremor which sometimes gave way for a time to a twitching of the muscles. The pupils were somewhat more dilated than normal. The muscles were definitely hypertonic. The animal had a wild frightened appearance.

Dec. 11: The heart rate was 96, the temperature 101.2 F. A slight tremor still remained, and the muscles were still fairly hypertonic. The animal was killed and the brain removed for study.

Lesion.—The needle passed along the caudal side of the optic tract at a level slightly cephalic to the mammillary bodies, and the solution was injected against the basal surface of the tuber cinereum. A part of the solution spread over the surface of the tuber cinereum forming a coagulated brown mass among the plexus of vessels and membranes at this point. Some of the fluid penetrated the brain substance, producing a large destructive lesion which began slightly cephalic to the mammillary body and extended caudad along the lateral side. The lesion was broad at the base and narrowed dorsally. Extending caudally and laterally, the lesion spread through the medial part of the cerebral peduncle, causing considerable damage. This lesion caused a total destruction of the most caudal cells of the nucleus tuberomammillaris and nucleus tuberis lateralis and some scattered cells of the substantia grisea. Anterior to this large lesion there was slight injury evident to the more superficial cells of all of these nuclei all along the basilar surface of the tuber cinereum on the left side.

Dog 21 A.—November 28: At 11:15 a. m., 0.1 cc. of a 0.3 per cent solution of mercuric chloride was injected into the region of the tuber cinereum through the left parietal lobe.

Symptoms.—At 2 p. m., the dog stood with the body and head bent to the left with the head hanging. The appearance and reactions suggested a demented condition. The pupils were almost completely dilated. There was a quantity of frothy saliva on the bottom of the cage. The muscles were hypertonic, especially on the left side. The temperature was 102.2 F. and the heart rate 150. Two moderately severe convulsions occurred within the hour, involving only the fore part of the body.

At 4:15 p. m., the animal lay in a half reclining posture against the side of the cage. The legs were extended and extremely rigid. The front legs were more spastic than the hind legs. The muscles of the neck and body were also spastic. Four convulsions occurred during the next twenty-five minutes. With these there was the usual frothing at the mouth, dilatation of the pupils and vasoconstriction. There was a twitching of the face and jaw muscles, but the remainder of the musculature of the body remained spastic during the seizure and did not pass into clonic spasm. The animal was in a state bordering closely on coma. In the interval between spasms the heart rate was 105 and the temperature 103.6 F.

At 4:40 p. m., the animal lay on the side in coma. The right legs were extended rigidily and strongly resisted bending. The left legs were hypertonic and extended, but were drawn up to the body when light pressure was placed on them or the foot was jerked. The muscles of the body and neck were hypertonic.

At 5:30 p. m., the animal had recovered sufficiently to walk with some staggering. There was a tendency to turn to the left. The animal seemed to be fairly well oriented.

November 30. The animal seemed rather stupefied. It either stood with the head drooping and the head and body bent to the left or walked in a small circle to the left in a blind, mechanical sort of way. The dog was clumsy and awkward. It walked over objects without noticing them, and when it walked into a corner, it had great difficulty in getting out.

December 1. At 3 p. m., the animal was in deep coma and appeared to be near death. There was an extreme rigidity in all muscles.

December 2. At 9 a. m., the animal was sitting in a normal position and was normal in appearance. At 4 p. m., the animal was again found to be lying on its side in rigidity and coma. The heart beat was weak; the rate was 144. The temperature was 101 F.

December 7. The animal was greatly emaciated. It appeared rather demented, but had a normal attitude of the body when at rest. During activity, the head and body were bent to the left, and the animal walked in a circle to the left in a blind, dazed manner. It staggered when walking, and the movements were slow, unsteady and inaccurate. The right legs were more affected than the left. The temperature was 102.6 F. and the heart rate 126.

Lesion.—The needle passed obliquely mediad and slightly cephalad through the thalamus, along the medial side of Forel's field and into the medial side of the internal capsule at a level slightly caudal to the optic chiasm. A part of the fluid passed upward and spread cephalad and caudad through the thalamus. The result was a large lesion in the anterior nucleus of the thalamus and the adjacent part of the lateral nucleus. A second part of the fluid spread through the medial third of the cerebral peduncle. Some of the mercuric chloride passed into the region of the fornix giving a lesion in the area dorsal and lateral to the fornix and extending nearly the entire length of the tuber cinereum. A small spur of the lesion in the peduncle passed medially along the base of the tuber cinereum slightly caudal to the chiasma. As a result of these two lesions in the tuber cinereum

there would be considerable injury and destruction to each of the three nuclei of the tuber cinereum.

Dog 22 A.—On December 5, at 9 a. m., 0.1 cc. of a 0.3 per cent solution of mercuric chloride was injected through the left parietal region.

Symptoms.—At 8 p. m., the animal was observed to have a severe convulsion which was concerned largely with the fore part of the body. The attack left the animal confused and disoriented. A second convulsion occurred thirty minutes later. The temperature at this time was 102.1 F., the heart rate 105.

December 6. At 1:35 p. m., the animal had a severe seizure. The body first became rigid; then a spasmodic twitching of the muscles began at the head and progressed back until the entire body was in clonic spasm. The animal fell on its side. During the remainder of the attack groups of muscles would frequently change intermittently from clonic to tonic spasm. During the convulsion the pupils were dilated, and there was frothing at the mouth. Several of these spasms followed at intervals of three minutes.

December 7. At 11:45 a. m., the animal was observed to have three severe convulsions at five minute intervals. In each of these there was a period of spasticity followed by tetany. The animal became unconscious and fell on its side. Salivation, dilatation of the pupils, vasoconstriction, erection of the penis and increase in heart rate were evident in each instance. Near the termination of two of these attacks a quantity of watery feces was passed with considerable force. After cessation of the muscular spasms the animal would lie for a few minutes in coma, breathing rapidly. It would then struggle to its feet and then run, flounder and fight its way around the cage as though it were having an hallucination. The animal would then gradually regain its orientation and appear to be normal. Another attack occurred about thirty minutes later, in which the rigidity phase was observed to last one and one-half minutes and the clonic phase forty-five seconds.

December 8. At 1:30 p. m., a severe seizure occurred. In this instance the rigidity lasted for sixty seconds and the clonic spasm for fifty seconds. The heart rate immediately following the spasm was 168. This fell to 120 within a few minutes after the attack. Nearing the end of the convulsion, the dog urinated and passed a large quantity of watery feces. A convulsion followed at 1:45 p. m. and another at 2 p. m. In the second case the rigidity and tetany phases each lasted for one and one-half minutes. Following the convulsion, the dog vomited a quantity of mucous-like substance and passed watery feces. The temperature following the spasm was 102.3 F.

December 9. The animal was cross and irritable and had the appearance of fright. The pupils seemed to be dilated more than normal. The temperature was 101 F.; the heart rate, 102. This condition existed without the occurrence of any additional convulsions for the next five days.

On December 14, the animal was killed and the brain removed for study.

Lesion.—The needle passed through the inferior horn of the lateral ventricle, thence ventral to the optic tracts to reach the tuber cinereum. A part of the mercuric chloride solution entered the lateral ventricle during the passage of the needle, while the remainder was injected against the base of the tuber cinereum. No gross lesion was evident in the tuber cinereum in this case, but an area along the base of the tuber cinereum was clearly affected by the solution injected. The supporting tissue in this area had the appearance of being coagulated into a network of coarse threads, leaving clear spaces between. The cells were shrunken and abnormal in appearance and occupied only a part of the cell spaces in which they lay. Many cells of the substantia grisea, nucleus tuberis lateralis and nucleus

tuberomammillaris were affected. The pia mater along the surface of the tuber cinereum was also somewhat broken up by the action of the mercuric chloride. There was considerable destruction in the walls of the inferior horn of the lateral ventricle.

Dog 24 A.—The animal was operated on on December 5, at 11 a. m., 0.1 cc. of a 0.3 per cent solution being injected into the region of the tuber cinereum through the left parietal lobe.

Symptoms.—Excessive salivation began soon after the operation. An hour after the operation, the animal began to have a series of rather mild spasms at intervals of about five minutes. Seven of these seizures were observed, the later ones becoming somewhat more severe. At this time the heart rate varied from 132 to 156, the heart beat at times becoming irregular. The temperature was 101.4 F. The seizures were limited almost entirely to the fore part of the body, and as is usually the case with the mild form of convulsion, were confined largely to clonic spasms with rigidity seldom appearing. The muscular symptoms usually began as a twitching of the facial muscles, then the jaw muscles, and then continued to progress back over the body. There was excessive salivation and dilation of the pupils. The animal became confused and somewhat dazed during the attacks but did not lose consciousness or fall. Two hours later, the animal was still observed to be having mild spasms.

At 8 p. m., the animal had a severe seizure in which it became extremely rigid and fell on the side unconscious. The tonic spasm soon gave place to a clonic spasm. It took some time for the dog to become oriented following the seizure. The heart rate at the end of the spasm was 216 and the temperature 101.6 F.

On December 6, at 2 p. m., the dog was found to be having severe convulsions following each other at intervals of from three to four minutes. The animal, at first dazed, soon passed into complete coma. The animal was then prepared for a study of the heart rate and blood pressure in relation to the convulsions. A cannula was placed in the carotid artery and connected to a mercury manometer which was arranged to write on a smoked drum. A second writing lever was attached by a string to the animal's front limb, just back of the elbow joint, in order to record the muscular spasms. The base line was marked off to show thirty seconds' intervals. Writings were made on both a slow and a rapid drum (fig. 4).[9]

9. A description of the kymograph record will be given under the discussion of the relationship of blood pressure and heart rate to the convulsions.

Lesion.—The needle passed along the ventral side of the left cerebral peduncle to reach the base of the tuber cinereum at a level cephalic to the mammillary bodies. The solution of mercuric chloride was injected against the base of the tuber cinereum and spread through the dense plexus of blood vessels and membranes in that region. A small amount of fluid passed to the opposite side of the tuber cinereum. A mass of brown coagulate substance lay along the entire base of the tuber cinereum on the left side, while a smaller mass lay along the base of the caudal end on the right side. The injury in this case was to the nerve cells and tissues lying near the periphery at the base of the tuber cinereum on the left side and to a small degree on the right side. In places the pia mater was broken up, and there were small softened areas penetrating the brain. The supporting tissue in many instances appeared as though it had been precipitated in heavy strands with spaces between. Injury to the nerve cells was evident along the entire extent of the periphery of the tuber cinereum on the left side and through the caudal end on the right side. The injured cells were scattered among cells which apparently had not been affected. Some cells appeared as shrunken

dark-staining bodies, while others showed more or less typical chromatolysis. The nucleus tuberomammillaris was subject to greater injury than either of the other nuclei of the tuber cinereum because of the large groups of nerve cells belonging to this nucleus which lie near the base of the brain.

Dog 25 A.—The animal was operated on on December 5, at 11:40 a. m., 0.1 cc. of a 0.3 per cent solution of mercuric chloride being injected through the left parietal region.

Symptoms.—A few minutes after the operation, the animal became rigid with the head thrown back and the pupils dilated. The rigidity soon gave place to a twitching of the muscles of the body accompanied by excessive salivation.

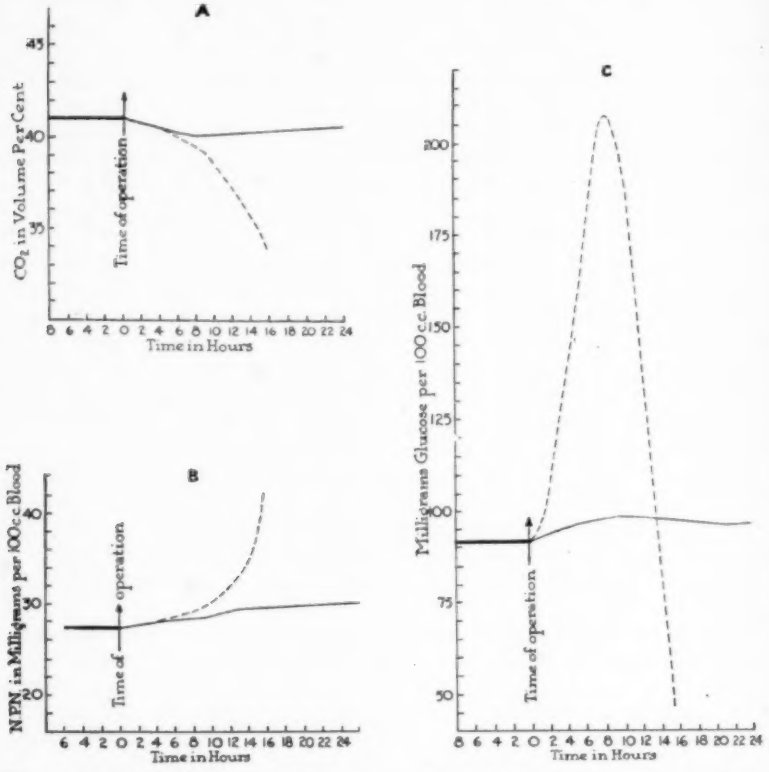

Fig. 3.—Graphs with compositive curves showing the trend of carbon dioxide combining power (*A*), nonprotein nitrogen (*B*), and dextrose (*C*), in the blood of dogs with lesion in the tuber cinereum, and in the control animals. The heavy solid line represents the preoperative average normal value. The broken line indicates results for animals with lesions; the light solid line, those for animals without lesions (controls).

At 12:15 p. m., the animal sat on its haunches driveling saliva. The pupils were dilated.

At 1 p. m., the heart rate was 114 and the temperature 102.2 F. The animal was noted to have an attack of violent tremor accompanied by excessive salivation and dilation of the pupils.

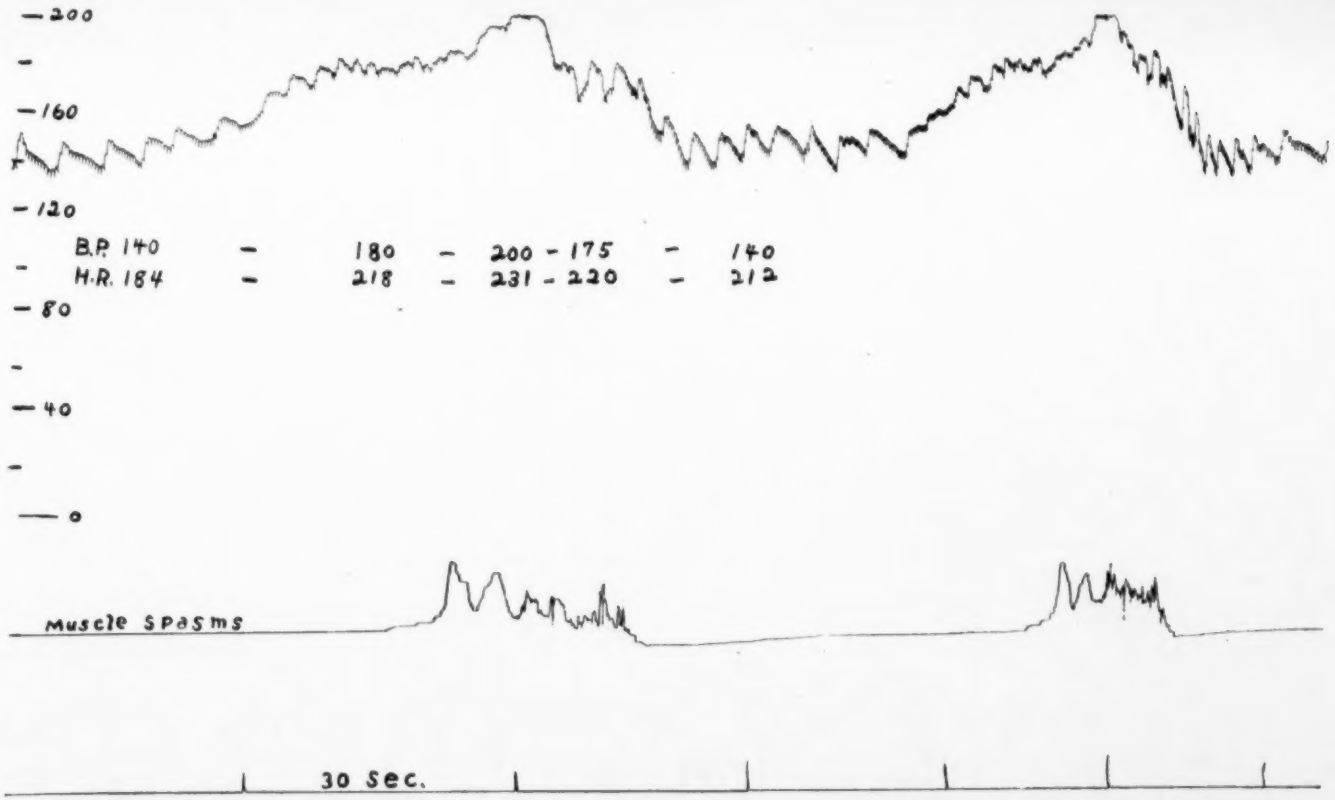

Fig. 4.—Kymograph record showing the blood pressure of a dog during convulsions and the relationship of blood pressure and heart rate to the muscular spasm.

At 8 p. m., the animal stood in a hypertonic, strained attitude with constant slobbering. There was an occasional mild twitching of the muscles of the head and face. The heart beat was irregular and at the rate of 130 per minute. The temperature was 104.

Beginning at 9 p. m., for about twenty minutes the animal had almost one continuous mild convulsion involving primarily the fore part of the body. The muscles of the face and jaws twitched constantly, while the fore limbs were sometimes spastic and sometimes in clonic spasm. During this time there was constant salivation and dilation of the pupils, but the animal did not completely lose consciousness or fall. This condition increased in intensity until the animal became helpless and fell on its side in a severe convulsion involving the entire body. Immediately following this attack, the temperature was 105 F. and the heart rate 288. The animal remained confused and frightened in appearance for some time.

At 9:35 p. m., the body had passed into a continuous state of rigidity, while the head and neck were in almost constant clonic spasm. The animal passed into complete coma with dilated pupils and excessive salivation. A cannula was placed in the carotid artery and connected with a kymograph in order to record the blood pressure and heart rate. The dog showed no signs of pain during the operation. The heart rate was found to be 250 per minute, and the blood pressure fluctuated between 150 and 190 mm. of mercury. Fifty-five cubic centimeters of blood was then drawn, after which the blood pressure slowly fell until at the end of ten minutes it registered 100 mm. of mercury, while the heart rate was then 280. The animal was considerably revived following the drawing of the blood. The heart beat became weaker, and the animal died several minutes later.

Lesion.—The needle passed ventral to the cerebral peduncle to enter the lateral side of the tuber cinereum at the anterior end of the mammillary bodies. A gross lesion extended from slightly in front of the mammillary body, passed caudal lateral to this structure and passed through the medial part of the subthalamic nucleus of Luys and the substantia nigra. The result was a destruction of the most caudal cells of both the nucleus tuberomammillaris and the nucleus tuberis lateralis, as well as some of the more scattered cells of the substantia grisea. The cells of all three of these nucleai which lay in the areas anterior and medial to the lesions were affected by the mercuric chloride, although not completely destroyed. There was also considerable cell destruction and injury in the subthalamic nucleus of Luys and the medial part of the substantia nigra.

Dog 28.—On January 7, at 9:30 a. m., 0.1 cc. of a 0.2 per cent solution of mercuric chloride was injected into the region of the tuber cinereum through the parietal region of the brain.

Symptoms.—At 1:25 p. m., there was a brief minor attack involving only the fore part of the body. This was followed one minute later by a severe and prolonged seizure. In the latter case the muscles of the body became suddenly spastic, with some tremor, and the animal fell on its side. The rigidity soon gave place to irregular convulsive contractions of the muscles, causing a jerking or twitching of all parts of the body. In addition to these spasmodic contractions which involved small groups of muscles, there was much clawing and thrashing about with the limbs and head. For a time the jaws snapped open and shut, then remained open in rigidity for a few seconds, following which they remained rigidly closed during the latter part of the seizure. During this attack there was excessive salivation, vasoconstriction, dilation of the pupils, erection of the penis and unconsciousness. The animal remained unconscious for about ten minutes following the seizure and was somewhat dazed and disoriented for twenty minutes more.

At 1:45 p. m., the temperature was 102.6 F. and the heart rate 108. Beginning at 2 p. m., there was a series of moderate convulsions at intervals of about five minutes. Another severe seizure such as the one already described occurred at 3 p. m.

At 4:20 p. m., the animal was in coma and convulsions were occurring every few seconds. The temperature was 109.1 F. and the heart rate 210. The convulsions at the later stage were much briefer in duration (from thirty to sixty seconds) and were characterized almost entirely by clonic spasms of the muscles, the tonic phase being absent. There was continuous frothing at the mouth and dilation of the pupils. The abdominal wall was opened and the viscera examined. The intestines were moderately dilated but not extremely flaccid. The stomach was flaccid and contained a small amount of sour-smelling, mucus-like substance. There was no peristalsis in either the stomach or the intestines. The bladder was moderately contracted. The gallbladder was distended and flaccid. The animal died soon after this examination was made.

Lesion.—The needle passed along the lateral side of the cephalic end of the midbrain, thence obliquely forward through the optic tract to reach the subarachnoid space at the base of the tuber cinereum. The mercuric chloride was injected against the basilar surface of the tuber cinereum at its caudal end and spread forward along both sides. The result was a slight injury to many of the more superficial cells along the ventral surface of the tuber cinereum on both sides. In a few places a small amount of the fluid penetrated the tuber cinereum for a short distance, causing small, well defined lesions reaching into the deeper parts of the nucleai. There was little total cell destruction and that was limited to the central part of the small penetrating lesions. There was marked injury, however, to many cells belonging to each of the three nuclei of the tuber, substantia grisea, nucleus tuberis lateralis and nucleus tuberomammillaris.

Dog 34 A.—The animal was operated on on February 19, at 10:30 a. m., 0.1 cc. of a 0.2 per cent solution of mercuric chloride being injected through the left temporal region.

Symptoms.—At 2 p. m., the animal was observed to be hypertonic and was having moderately severe convulsions at intervals of from one to five minutes. These involved largely the fore part of the body. Rigidity at the onset was followed by clonic spasms characterized by snapping of the jaws, spasmodic jerking of the head and a twitching of the musculature of the anterior part of the body. During this time there was frothing at the mouth, vasoconstriction, erection of the penis, dilation of the pupils, and urine was ejected by a series of small forceful spurts.

These moderate convulsions were followed a few minutes later by a severe attack lasting for over two minutes. The animal appeared frightened and disoriented; this was followed almost instantly by a dilation of the pupils, salivation, vasoconstriction and a stiffening of the entire body. The animal plunged forward on its head and fell on the side in a state of extreme rigidity. The rigidity was followed after a few seconds by clonic spasms. There was a snapping of the jaws, jerking of the head, and a twitching of all the musculature of the body. During this time the animal was apparently unconscious and insensible to pain or outward stimuli. A quantity of watery feces was discharged with a great deal of force, and the dilation of pupils, frothing at the mouth, vasoconstriction and erection of the penis persisted throughout the attack. For thirty minutes following this attack, the animal lay on its side in a dazed, semicomatose state with the occasional occurrence of light convulsions similar to those already described.

At 3:25 p. m., the heart rate was 264 and the temperature 101.2 F. Five severe convulsions occurred within the next thirty minutes. These severe attacks continued then at intervals of a few seconds, each convulsion lasting for from one and one-half to two minutes. The animal did not completely recover consciousness between the spasms. At 4:20 p. m., the temperature had risen to 105 F. Respiration was much interfered with during the spasm, and the animal would pant rapidly during the intervals.

At 6:30 p. m., the temperature of the animal was 109 F. It was in a state of complete coma, and the convulsions were almost continuous. The heart beat was weak and too rapid to be counted. The abdominal wall was opened without the animal showing any reaction of pain. The intestines and stomach were observed to be relaxed, and the bladder was in a state of moderate contraction. The animal died several minutes later.

Lesion.—Microscopic examination of the brain showed that the needle passed ventral to the left optic tract and entered the plexus of blood vessels and membranes lying at the base of the tuber cinereum, the fluid being injected against its surface. A mass of light brown coagulated substance lay along the base of the tuber cinereum. The fluid penetrated in a number of places into the lateral side of the tuber cinereum, causing injury to groups of cells belonging to the nucleus tuberis lateralis, nucleus tuberomammillaris, and the more scattered cells of the substantia grisea. Many nerve cells in this area were in the process of degeneration with frequent neuronophagia. Many cells in the more medial basilar portion of the substantia grisea showed chromatolytic changes of a varying degree, but there was not the gross injury here which was present more laterally. Some of the fluid leaked into the inferior horn of the lateral ventricle to which the needle passed in close proximity, causing considerable injury in the walls of the ventricle at this level.

Dog 40 A.—The animal was operated on at 10:30 a. m., through the lower temporal region as described by Cushing. Two injections of 0.1 cc. of a 0.3 per cent solution were made in the region of the tuber cinereum.

Symptoms.—At 2 p. m., the heart rate was 240, the temperature 107.7 F. and the rate of respiration 90 per minute. The pupils were almost completely dilated, and the animal was in a state bordering on coma.

This condition continued essentially the same throughout the remainder of the day until the animal died at 7 p. m.

Lesion.—In the first lesion the needle entered the right side immediately caudal and lateral to the optic chiasm and continued obliquely upward through the medial side of the internal capsule. There was little injury to the internal capsule since the fluid spread forward through the base of the frontal lobe. There was some injury to the basal optic ganglion.

In the second lesion the needle entered the base of the tuber cinereum caudal to the optic chiasm and just to the left of the third ventricle. The needle then passed obliquely upward through the third ventricle and the upper half of the wall of the ventricle on the opposite side. Some fluid leaked into the ventricle and caused some injury to the cells in the immediate wall of the ventricle. The lesion which followed the needle was confined to a narrow zone extending through the ventral part of the left ventricular wall and the dorsal part of the right ventricular wall. In either case the lesion lay close to the third ventricle. As a result there was much injury with little total destruction in the cells of the substantia grisea. There was probably little injury to the other two nuclei of the tuber cinereum.

Dog 43 A.—On December 7, at 11:45 a. m., 0.1 cc. of a 0.5 per cent solution of mercuric chloride was injected into the region of the tuber cinereum through the left parietal lobe of the brain.

Symptoms.—At 2:30 p. m., the heart rate was 104 and the temperature 103.2 F. There was moderate erection of the penis, vasoconstriction was indicated and the pupils were dilated to about twice the normal size.

At 8 p. m., and again at 8:25 p. m., the animal had a fairly severe seizure which involved largely the fore part of the body and did not result in a complete loss of consciousness. The pupils remained constantly dilated at this time, and the animal was restless and hypertonic. There was the usual salivation, vasoconstriction and partial loss of consciousness which accompany light spasms. Following the first of these seizures the heart rate was 192. Within a few minutes this had dropped to 112.

At 8:30 p. m., there was a severe and prolonged seizure involving complete loss of consciousness. The animal fell on its side and passed through both the tonic and the clonic phases of the convulsion. The seizure was followed for a few minutes by the animal remaining on its side and thrashing the air with its feet in a manner which resembled awkward, disorganized running movements. The animal remained disoriented for some time following the cessation of these involuntary movements. Another such convulsion occurred twenty minutes later. At this time it was noted that the eyes protruded more than normally.

On December 8, the animal was found dead in the cage.

Lesion.—The lesion in this case involved practically the entire tuber cinereum on the left side and caused considerable injury on the right side. The lesion was a large one, beginning in the head of the corpus striatum, shifting toward the midline and continuing caudad to the mammillary bodies. Throughout the extent of the tuber cinereum the lesion occupied practically the entire area occupied by the three nuclei of the tuber cinereum. The lesion was not solid, however. There were irregular patches of tissue in which destruction was complete, and interspersed between these patches were areas of cells that showed severe injury but not complete destruction. There was thus perhaps total destruction of half of the total area of the tuber cinereum on the left side. Some fluid passed into the third ventricle and caused considerable injury to the more superficial cells bordering the ventricle on the right side.

Dog 50 A.—On December 11, at 11:30 a. m., 0.1 cc. of a 0.3 per cent solution of mercuric chloride was injected into the region of the tuber cinereum by way of the upper parietal region of the brain.

Symptoms.—At 2:55 p. m., the animal appeared tense and irritable. The eyes seemed to bulge and slight vasoconstriction and dilation of the pupils were evident. The dog was having light and moderately severe convulsions at intervals of from five to ten minutes. These involved mainly the fore part of the body, and the animal did not completely lose consciousness or fall. The heart rate was 156 and the temperature 103.4 F.

At 4:45 p. m., the animal was badly disoriented and was having severe convulsions at brief intervals. These symptoms continued to increase in severity during the next hour until the animal passed into a state of complete coma with convulsions occurring at intervals of a few seconds. These were the typical convulsions involving tonic and clonic spasms, although in this later stage the clonic spasms predominated. There was excessive salivation, complete dilation of the pupils, vasoconstriction and an increase in heart rate. The rate of heart beat at this time was 288 and the temperature 111 F. The animal died about 5:45 p. m.

Lesion.—The needle passed ventrally and slightly cephalomediad through the lateral side of the anterior nucleus of the thalamus and the medial side of Forel's field to reach the dorsal part of the tuber cinereum. There was a narrow slitlike lesion following the path of the needle through the thalamus. At its ventral end the lesion spread out into a fairly broad, softened area involving the medial side of Forel's field and the dorsolateral part of the tuber cinereum at a level cephalad to the mammillary bodies. This resulted in the destruction of and injury to many cells of the substantia grisea and the more dorsal cells of the nucleus tuberomammillaris, with probably little or no injury to the nucleus tuberis lateralis.

Dog 51 A.—On January 2, at 9:30 a. m., 0.1 cc. of a 0.3 per cent solution of mercuric chloride was injected into the region of the tuber cinereum by way of the upper parietal region of the brain.

Symptoms.—Throughout the first day and the early part of the second the animal was apparently normal. The temperature taken at 1:45 p. m. on the first day was 104.6 F., and the heart rate was 108.

At 4:30 p. m. on the second day, the animal was found in a dazed condition, with severe convulsions occurring at brief intervals. These were the typical seizures involving unconsciousness, muscular spasms, dilation of the pupils, vasoconstriction, excessive salivation and increased heart rate. The convulsions continued to increase in severity and frequency until the animal was in continuous coma with the spasms occurring at intervals of a few seconds. The dog died at 5:30 p. m. In this case the seizures commenced rather suddenly, several hours after the operation, and progressed rapidly toward the status epilepticus stage and death.

Lesion.—The needle passed through the central region of the thalamus, along the medial side of Forel's field, and into the region of the fornix slightly cephalad to the mammillary bodies. A rather broad lesion extended along the course of the needle through the thalamus, the medial side of Forel's field and the region of the fornix. The ventral end of the lesion was confined to about the caudal half of the tuber cinereum. At this level the lesion destroyed the region dorsal and lateral to the fornix and a narrow zone around the ventromedial side of the fornix. Of the region which is occupied by the three nuclei of the tuber cinereum there was a destruction of about the dorsolateral half, beginning at the mammillary body and extending forward through about one-half the extent of these nuclei. This would result in injury and destruction of a great many cells of each of the nuclei located in this region (substantia grisea, nucleus tuberis lateralis and nucleus tuberomammillaris) with the nucleus tuberis lateralis probably suffering less injury than the other two.

BLOOD CHEMISTRY

It is apparent from a review of the literature, as well as from the proximity of the hypophysis to the tuber cinereum, that much of the information concerning the functions of the latter has been obtained by chance lesions in this center when working on the pituitary. Thus Erdheim[10] was the first to postulate that disturbance in the centers of the hypothalmus is the essential cause for many symptoms attributed

10. Erdheim: Sitzungsb. d. k. Akad. d. Wissensch. Math.-naturw. Cl., Wien **113**:537, 1904.

to the hypophysis. Later, Aschner [11] expressed the same opinion. To interpret clinical observations or metabolic irregularities on such data, from either points of interest, it is requisite to know histologically whether one or both of these areas are involved. Experimentally, it seems to be well established that extensive injury to the tuber cinereum is incompatible with life.

So far as blood chemistry is concerned, our studies were limited to changes in calcium, potassium, nonprotein nitrogen, urea, and dextrose content of the blood. A few observations were made on the carbon dioxide combining power of the plasma. Special effort was made to follow the same technical procedure in the quantitative determination of each of these constituents in order to obtain comparable results. The blood sample was always obtained by cardiac puncture. Normal values were based on samples taken at least twelve hours after the last feeding. This is particularly important in defining the normal blood sugar level. The animals which did not develop the typical symptoms and on histologic examination showed no lesion in the tuber cinereum were used as controls and were, for our purpose, considered normal.

In view of the fact that dogs with lesions in the tuber cinereum showed muscular spasms which in some respects resembled tetany, it was thought that it might be due to a change in the calcium or potassium content of the serum, or a shift in the potassium: calcium ratio. The method used for the determination of calcium was essentially the Clark-Collip [12] modification of the Kramer-Tisdall method,[13] for potassium, the Kerr modification of the Kramer-Tisdall method.[14] The results obtained are summarized in the accompanying table.

From a study of the figures in the table one can conclude nothing definitely enough to cause one to assume that the muscle spasms resembling tetany are caused by a diminution in serum calcium or a change in the potassium: calcium ratio. True, there are more low figures in the calcium column on the left and a greater discrepancy between high and low values as compared to the control calcium column on the right.

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13. Kramer, B., and Tisdall, F. F.: A Simple Technique for the Determination of Calcium and Magnesium in Small Amounts of Serum, J. Biol. Chem. **47**:475, 1921.

14. Kerr, S. E.: Studies on the Inorganic Composition of the Blood. I. The Effect of Hemorrhage on the Inorganic Composition of Serum and Corpuscles, J. Biol. Chem. **67**:689, 1926.

However, owing to the limited number of observations and the experimental error involved, no conclusions seem warranted. Even if we were dealing with tetany of the usual type, it would be erroneous to assume that it is necessarily accompanied by, or due to, hypocalcemia. It is of interest at this point to recall that some investigators [15] have observed a slight decrease or no change in serum calcium in cases of epilepsy. Reiter [16] has even reported high calcium figures. From a broader point of view, changes in serum calcium have no relation to motor disturbances (irritability) in manic-depressive diseases.[17]

The technical procedure used for the determination of the carbon dioxide combining power of the blood was that described by Van Slyke and Neill [18] for the manometric type of apparatus. Our observations

Potassium and Calcium Data on Dogs Which Showed Definite Lesions and Symptoms and on Control Animals

	Values Expressed in Mg. per 100 Cc.			Control Animals			
Animal	Calcium	Potassium	Potassium/ Calcium	Animal	Calcium	Potassium	Potassium/ Calcium
3	14.2	38.3	2.70	2	16.6	35.9	2.16
8	12.45	29.1	2.34	4	13.7	27.5	2.01
8a	12.4	38.1	3.07	5	11.8	23.3	1.97
9	14.2	20.7	1.46	6	11.9	23.0	1.93
9a	16.3	27.7	1.70	7	11.3	22.4	1.98
19	9.9	17.9	1.81	10	11.6	24.3	2.10
19a	10.1	20.9	2.07	11	9.9	23.9	2.41
20	9.5	20.8	2.19	12	13.2	25.7	1.95
21	10.3	16.1	1.58	13	11.9	22.2	1.87
21a	11.0	24.0	2.18	14	11.7	19.3	1.65
22	6.7	22.2	3.32	16	12.4	17.2	1.38
22a	9.9	13.1	1.32				
25	13.8	16.1	1.17				
Average	11.6	23.46	2.02		12.36	24.06	1.94

15. Jansen, W. H.: Kalkstudien am Menschen (III. Mitteilung). Der Kalkgehalt des menschlichen Blutes unter pathologischen Verhältnissen, Deutsches Arch. f. klin. Med. **144**:14, 1924. Hamilton, Bengt.: A Comparison of the Concentrations of Inorganic Substances in the Serum and Spinal Fluid, J. Biol. Chem. **65**:101, 1925. Salvesen, H. A.: Blodkalken under normale og visse patologiske Tilstande, Norsk mag. f. laegevidensk. **84**:1047, 1923. Hendriksen: Ca i blod og spinalvaedske hos epileptikere, Neurologisk Selsk. Förhandl. Hospitalstid. **68**: 52, 1925.

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17. Weston, P. G., and Howard, M. Q.: The Determination of Sodium, Potassium, Calcium, and Magnesium in the Blood and Spinal Fluid of Patients Suffering from Manic-Depressive Insanity, Arch. Neurol. & Psychiat. **8**:179 (Aug.) 1922. Tómasson, Helgi: Undersøgelser over nøgle af blodets electrolyter og det vegetative nervensystem saerlig hos patienter med manio-depressive psykose, Copenhagen, Levín & Munksgaard.

18. Van Slyke, D. D., and Neill, J. M.: The Determination of Gases in the Blood and Other Solutions by Vacuum Extraction and Manometric Measurement, J. Biol. Chem. **61**:523, 1924.

indicate that in animals with lesions in the tuber cinereum there is a condition of progressive acidosis; that is, as the frequency and severity of the fits increased, the carbon dioxide combining power of the plasma decreased. The results are shown in the form of composite curves in figure 3 A. From an objective point of view, the etiology of this condition is probably not singular. 1. Muscle spasms no doubt interfere with normal breathing and complete ventilation of the blood in the lungs. 2. Retention of nitrogenous constituents may play a rôle. 3. Hyperglycemia caused by the release of stored sugar may lead to incomplete oxidation with the formation of acid end-products, or the mere presence of more dextrose may render the blood incapable of holding the normal quantity of carbon dioxide.

As already indicated, a mild retention of nitrogen, which in rare cases bordered on uremia, was observed. In the case of the nonprotein nitrogen value this retention appeared to be progressive until the animal died. The urea-nitrogen values lacked definiteness but in some cases ran parallel with the nonprotein nitrogen values. Because we have experienced wide variations in the figures for both nonprotein and urea nitrogen on apparently normal dogs, it is doubtful whether much significance can be attached to these results. The average of the nonprotein nitrogen curves for both pathologic and control animals is shown in figure 3 *B*.

In our studies, blood sugar changes following lesions in the tuber cinereum were by far the most definite. Invariably the sugar content rose rapidly to about twice the normal value, at which time the epileptic-like syndrome was well established; then, as the fits became more frequent and more severe, the sugar content decreased rapidly until the animal died in a comatose condition. When shown graphically, as in figure 3 *C*, it presents a condition of progressive hyperglycemia which reaches a maximum from six to eight hours after operative injury, followed by a rapid disappearance of dextrose from the blood. Sugar values a few minutes before death were often one-half the normal figure. The blood sugar curve of the control animals presents a different picture.

The fact that the rise in blood sugar occurs before muscular spasms become severe would seem to indicate that the cause cannot be explained wholly on the basis of muscular activity. Other mechanisms are no doubt involved. Hyperactivity of the adrenal glands probably plays the major rôle. The downward trend of the sugar curve represents rapid utilization of dextrose by a spastic musculature.

In the course of our experiments we examined the livers of three animals with lesions in the tuber cinereum in order to ascertain the degree of depletion of the carbohydrate reserves. The absence of glycogen in the livers of these animals indicated the complete mobiliza-

tion of this carbohydrate in the blood stream. It may be well to emphasize that the period of inanition of these animals is not long enough to exhaust the carbohydrate reserves. In animals in which we failed to injure the tuber cinereum (controls) but caused enough disturbance to bring about a condition of anorexia, we have observed that even after three days considerable glycogen was present in the liver.

Camus and Roussy [19] punctured the hypothalamus of dogs with a heated drill and produced a transient polyuria. They concluded that polyuria was due to a lesion in the gray matter of the tuber cinereum in the region of the infundibulum. Later, the same authors [20] showed that polyuria does not depend on a lesion in the hypophysis by producing it in animals after hypophysectomy. Houssay [21] reported similar results.

Bailey and Bremer,[22] using a slightly different technic, confirmed the observations of Camus and Roussy that hypothalamic injury caused polyuria and pointed out further that, depending on the extent of the lesion, the symptom may be transient or permanent. When permanent polyuria was produced, it had all the characteristics of diabetes insipidus in man. Glycosuria, in their experience, was an inconstant result and in their opinion depended on the state of nutrition of the animal.

Leschke [23] concluded that injury to the tuber cinereum rather than a disturbance of the pituitary is the cause of diabetes insipidus.

Studies of metabolism in epilepsy with particular emphasis on the sugar level in the blood have been made and the literature reviewed by Lennox.[24] The data collected by him, from a subjective point of view, give no evidence of abnormality of carbohydrate metabolism that might in itself induce epileptic seizures. He concluded, "that increase of blood sugar during convulsions would depend on the presence of available glucose in the body." His argument that a rise in blood sugar

19. Camus, J., and Roussy, G.: Hypophysectomie et polyurie expérimentales, Compt. rend. Soc. de biol. **75** 483, 1913.

20. Camus, J., and Roussy, G.: Experimental Researches on the Pituitary Body. Diabetes Insipidus, Glycosuria, and those Dystrophies Considered as Hypophyseal in Origin, Endocrinology **4**:507, 1920; Diabète insipide expérimental et opothérapie hypopysaire, Compt. rend. Soc. de biol. **83**:1578, 1920.

21. Houssay, B. A.: The Pituitary Body and Polyuria, Endocrinology **2**:94, 1918; Sur la polyurie soi-distant hypophysaire, Compt. rend. Soc. de biol. **81**: 381, 1918.

22. Bailey, P., and Bremer, F.: Experimental Diabetes Insipidus, Arch. Int. Med. **28**:773 (Dec.) 1921.

23. Leschke, E.: Beiträge zur klinischen Pathologie des Zwischenhirns (I. Mitteilung). Klinische und experimentelle Untersuchungen über Diabetes, seine Beziehungen zur Hypophyse und zum Zwischenhirn, Ztschr. f. klin. Med. **87**:201, 1919.

24. Lennox, W. G.: Studies of Metabolism in Epilepsy, Arch. Neurol. & Psychiat. **18**:383 (Sept.) 1927.

may be attendant on any sudden muscular activity and not necessarily peculiar to an epileptic fit is, of course, logical.

Sachs and MacDonald [25] studied blood sugar values in hypophysectomized dogs following lesions in the hypothalamus. They observed polyuria and a transient glycosuria. Their observations on the trend of the blood sugar level coincide fairly well with ours. The outstanding difference is one of degree.

Di Renzo [26] reported some interesting observations on cerebropathic epileptic subjects. In ten cases examined during the epileptic seizure, he observed a constant but transient hyperglycemia. He stated that hyperglycemia in cerebropathic subjects is due to lesions of the brain.

Pagniez [27] reported that when he obtained serum from epileptic patients at the time of the seizure and injected it into the heart or carotid artery of animals, it produced a series of convulsions which lasted about half an hour and sometimes ended fatally. If this observation were repeated and controlled, it might seem to indicate that in epilepsy a toxic product is elaborated in the blood. To this end we obtained the serums of several dogs during severe muscular spasms and injected 50 cc. into the hearts of normal dogs. The results were entirely negative; we were unable to detect any change in blood sugar or abnormal behavior of the animals.

SYMPTOMATOLOGY

The convulsion we are dealing with following lesion in the tuber cinereum is a complex reaction involving several factors. The symptoms resolve themselves into two main groups: (1) the muscular spasm involving the voluntary muscles of the body and (2) the disturbance of functions that are under the control of the autonomic nervous system (salivation, dilation of pupils, etc.). These two sets of symptoms are combined in the typical seizure or convulsion. Except in those cases in which the symptoms were modified somewhat by the lesion involving the cerebral peduncle, subthalamic nucleus of Luys or some adjacent region of the brain, there is a striking uniformity in the symptomatology exhibited in this group of experimental animals. In the description of cases we have often referred to mild convulsions, moderate or fairly severe convulsions and severe convulsions. We shall discuss in a general way these three types of convulsions before attempt-

25. Sachs, E., and MacDonald, M. E.: Blood Sugar Studies in Experimental Pituitary and Hypothalamic Lesions, Arch. Neurol. & Psychiat. **13**:335 (March) 1925.

26. Di Renzo, F.: Researches in Glycemia. II. Glycemia in Epileptic Persons During the Seizures and During the Intervals, Arch. di. pat. e clin. med. **4**:411, 1925.

27. Pagniez, J.: Recherches sur la toxicité du sérum et du liquide céphalorachidien des épileptiques, Presse méd. **32**:45, 1924.

ing to analyze the convulsion and discuss the various individual symptoms which are combined into the typical seizure.

1. *The Mild Convulsion.*—The onset of the convulsion may occur any time from two to twenty hours after the operation. In the majority of cases they begin within from three to six hours. The first convulsions are nearly always of the mild type. At this stage the autonomic symptoms are much more pronounced than the muscular spasms. The pupils dilate, there is frothing at the mouth, the skin and oral mucosa become blanched and the heart rate is increased. The animal appears confused and disoriented. There is a snapping of the jaws, twitching of the face and more or less twitching of the muscles of the neck and shoulders, which occasionally spreads to the fore limbs. These attacks are usually of only from twenty to thirty seconds' duration, although they may sometimes last for a much longer period. The animal recovers rapidly from these mild convulsions. The frequency of these mild spasms and the length of time during which they occur before the onset of more severe convulsions vary considerably with individual cases. This variation is probably dependent on the extent of the lesion in the tuber cinereum. The mild convulsion may continue to occur in the intervals between more severe seizures after they appear.

Moderately Severe Convulsions.—In most of the animals studied the convulsions become progressively more severe and at the same time more frequent. In this course of events there is usually an intermediate period when the convulsions are more severe than the mild attacks described and yet they are not of the severe type which occur more often at a still later stage. These moderately severe convulsions are of longer duration (usually from thirty to ninety seconds). The autonomic symptoms are slightly more severe than in the mild spasm, and the animal appears considerably more confused and disoriented. The spasms involving the voluntary musculature of the body are much more severe and involve more or less the entire body. However, there is nearly always a tendency for the musculature of the fore part of the body to be affected most. There also seems to be a tendency for the spasms to develop first in the fore part of the body and then to spread to the remainder of the body. The behavior of the muscles in this type of convulsion suggests a state on the border line between tonic and clonic spasms. In most cases there is not discernible a well defined tonic spasm followed by a clonic phase, as is typical of the severe convulsion to be described. The musculature acts independently in smaller groups or within a restricted area of the body. Thus, a certain group of muscles may pass from tonic to clonic spasm and then return to the tonic condition several times during a convulsion. In other words, the behavior of the voluntary musculature does not conform to a definite pattern in this type of seizure, and a great deal of variation

is exhibited in this behavior. As the convulsions increase in severity, there is more and more a tendency for the muscles to pass first into tonic spasms and for this to change into a well defined clonic spasm such as that which is characteristic of a typical epileptic seizure of the grand mal type.

Severe Convulsions.—The third type of convulsion, which occurs more frequently at a still later stage, has been referred to as a severe convulsion or seizure and resembles closely the grand mal type of seizure which is characteristic of epilepsy in man. In this type of convulsion the animal loses consciousness and falls. The convulsion comes on suddenly in an animal which otherwise seems to be perfectly normal. In many instances it has been noted that if the animal is lying down, it will rise to its feet a moment before the spasm occurs and has the appearance of fright. If moving about, the animal has sometimes been seen to stop suddenly and present momentarily an appearance of fright and disorientation before the onset of the convulsion. We have attempted throughout this series of experiments to determine as far as possible the sequence in which various symptoms occur. The suddenness with which the seizure occurs and the severity of the symptoms from their onset make this a difficult question to determine. In several instances it has seemed that accompanying the brief period of fright which the animal has seemed to experience there is a dilatation of the pupils, a beginning of salivation and vasoconstriction. It is believed that the symptoms usually precede momentarily the appearance of the muscular spasm. In the severe seizure the dog becomes suddenly rigid over the entire body. The animal then plunges forward or falls on the side and lies for a time in extreme rigidity. The rigidity soon gives place to a spasmodic twitching of all the muscles of the body. The entire convulsion usually lasts from one and a half to three minutes, although it may occasionally be of longer duration. There is some variation in the length of time which the tonic and clonic phases of the spasm last. In the majority of cases the time is fairly equally divided between the two. During the convulsion the dog is unconscious, the pupils are completely dilated, vasoconstriction is evident and there is a marked increase in heart rate and excessive salivation. In a few cases in which the temperature was taken immediately before and after the convulsion there was a rise of about 1 degree F. or more in body temperature. Frequently, there was urination during the convulsion. Sometimes during or following a seizure a large amount of watery feces was passed with great force.

Although there is a tendency for the mild spasm to predominate at the onset of symptoms, the moderate spasm in the intermediate stage and the severe spasm to be more common at a later stage, it must not be supposed that these various types of convulsions are necessarily

confined to a certain period. A severe convulsion may sometimes occur near the onset of symptoms, or a mild convulsion may frequently occur at a later stage in the interval between more severe seizures. Furthermore, there is not a sharp line of distinction that can be drawn between the convulsions of varying severity. It is hoped merely that in recognizing the progression from a mild to a severe convulsion and in studying the characteristics of each one may be led to a better understanding of the nature of a convulsion, as well as of the function of the tuber cinereum.

In the majority of cases studied, the animal finally passed into a state which closely resembles what is known as status epilepticus in the human being. At this stage the animal is in complete and continuous coma. The heart rate usually increases to 250 or more. The temperature rises rapidly, usually reaching to from 108 to 111 F. In the two cases recorded the blood pressure was high. In the few instances in which the abdominal contents were examined, the stomach and intestines were relaxed and inactive, the bladder was empty and contracted but the muscles did not appear hypertonic. The animal is almost continually in muscular spasm, although the contractions are predominantly of the clonic type. The spasm continues for from thirty to sixty seconds, during which time the respiration is greatly interfered with, then the spasm almost or entirely ceases for a few seconds during which the animal pants rapidly. At the beginning of the spasm there may be a momentary rigidity of the muscles, but this is always of short duration. In the interval between spasms, the muscles remain hypertonic. At this time the animal does not react to pain or stimulation of any kind.

COMMENT

The question may well arise as to why other investigators doing experimental work in the region of the tuber cinereum have not noted convulsions in their animals? Many of these investigators have had in mind only one symptom, such as changes in blood sugar, polyuria, etc., and it is possible they have overlooked the convulsions, if present, or dismissed them as having no bearing on the question. On beginning experimental work on the tuber cinereum, we made a practice of operating in the afternoon and giving little attention to the animal until the following day. Several animals died during the night, and it was invariably found that these animals had a well placed lesion in the tuber cinereum. It was only when the animals were kept under close observation during the few hours following the operation that convulsions were noted. Bailey and Bremer recorded convulsions in the case histories of two of their animals and epileptic attacks in a third. However, they attached no significance to these symptoms. A second factor bearing on this question may have to do with the variety of

methods used in making lesions in the tuber cinereum. Destruction of a large area in the tuber cinereum would no doubt give different symptoms from those produced by introducing a reagent which might set up an irritation but cause little cell destruction.

We believe that in this series of animals we are dealing primarily with an irritative lesion. In that event the convulsion is to be interpreted as a result of an irritative stimulation of the nuclei of the tuber cinereum rather than as a result of loss of function. The combination of symptoms which follow a lesion in the tuber cinereum may be regarded as either a direct or an indirect result of a diffuse, unregulated discharge of nerve impulses from the cell groups of the tuber cinereum. Since this is not a normal regulated impulse, the symptoms will not represent a normal function of these centers, but will probably represent an exaggeration of the normal function. The lesion in these cases was made by injecting a weak solution of mercuric chloride into the region of the tuber cinereum. It will be noted that the symptoms were more pronounced in those cases in which the injected solution spread profusely through a large area in the tuber cinereum, and were less severe in those cases in which the lesion was a well constricted destructive one. In the former there was an injury to a large number of cells over a large area, while in the latter there was complete destruction of many with relatively little injury and irritation to cells in the area surrounding the lesion. It is also of interest to note that in those animals which recovered without passing into the "status epilepticus" stage, the convulsions occurred only during a period of from one to three days following the operation. This might be taken as further indication that the symptoms were due to a temporary irritation in the tuber cinereum rather than to a loss of function.

The three cell groups of the tuber cinereum (substantia grisea, nucleus tuberis lateralis, and nucleus tuberomammillaris) are so closely intermingled that it is practically impossible on the basis of localized lesions in the tuber cinereum to isolate the symptoms which may be ascribed to each. In an investigation now being conducted by Morgan it is hoped that certain drugs may be found which may have a selective action on these cell groups and provide a better means of determining the specific function of each of the nuclei of the tuber cinereum.

We believe that in the tuber cinereum we are dealing with cell groups that are to be considered as secretory centers for certain of the internal secreting glands, particularly the suprarenal, thyroid and parathyroid. This view is supported by some work by Morgan [28] which, owing to the small number of specimens studied up to the present time, has been published only in abstract form. In five dogs a successful

28. Morgan, L. O.: Further Observations on Mammillo-Infundibular Region of Diencephalon, Proc. Soc. Exper. Biol. & Med. **25**:617, 1928.

lesion was placed in the tuber cinereum. After convulsions had progressed to the stage where they were occurring at frequent and regular intervals, either the suprarenal and thyroid and parathyroid glands were removed or the blood vessels were ligated. It was found in these animals that removing or tying off the thyroids and parathyroids caused almost instant cessation of the muscular spasms. However, this had no effect on the remaining symptoms. On the other hand, tying off or removing the suprarenal glands caused the pupils to constrict to normal, the heart rate fell from 250 or more to less than 100, and the intestines and stomach recovered their tone and began peristalsis. The muscular spasms became slightly less severe but did not cease. Removing or clamping the glands on one side had a marked effect but did not stop the symptoms.

That group of symptoms which we have called the "autonomic" symptoms has been observed to occur without the rest. They include dilatation of the pupils, excessive salivation, vasoconstriction, increase in blood pressure, increase in the rate of heart beat and relaxation of the stomach and intestines. The occurrence of these symptoms always as a group would indicate that there is some basic mechanism back of all these disturbances. It is well known that this same group of symptoms may be obtained with injections of epinephrine. These are also the symptoms which we would expect to occur should there be a hyperactivity of the suprarenal glands.

There are many indications that the group of autonomic symptoms is a more important and basic factor in the convulsion than the muscular spasm. There is reason to believe that these symptoms begin slightly before the muscular spasm. While there is great variation in the muscular spasm, the autonomic symptoms are fairly uniform through all types of convulsions, becoming only somewhat more pronounced as one proceeds from a mild to a severe spasm. In many cases these symptoms continue to be present to a varying degree between the actual convulsions. Dilatation of the pupil, vasoconstriction and other autonomic symptoms continue for a time after the muscular spasm has ceased.

The relationship of the heart rate and blood pressure to the convulsion is shown in figure 4, which is based on a kymograph record made from dog 24. The blood pressure and heart rate were recorded by means of a cannula in the carotid artery. The muscular spasms were recorded by means of a string attached back of the elbow joint in the front leg. At the time the reading was taken, the animal was in coma, and convulsions were occurring at intervals of about forty seconds. Time was marked off on the base line at thirty second intervals. The convulsions are recorded on a slow drum; the second is recorded somewhat more slowly than the first. The average blood

pressure between convulsions is about 140 mm. of mercury. The average rate of heart beat in the intervals between convulsions is about 200 per minute. At the beginning of the change incident to a convulsion the blood pressure is seen to rise slowly to about 180 mm. This initial rise occupies about twenty seconds time. The blood pressure remains at this level for about eighteen seconds and is followed by a second and more rapid rise (ten seconds) to a pressure of 200 mm. The pressure then remains at this peak for from six to seven seconds, after which there is a rapid fall to about 175 mm. This fall in pressure is immediately followed by the pressure gradually returning to the original level of 140 mm. This final fall in pressure occupies a period of about fifteen seconds. The rate of heart beat is in general parallel to the blood pressure. In the interval preceding the first convulsion recorded, the heart rate was about 184. Corresponding to the first rise in blood pressure, the heart rate increases to 218; corresponding to the peak in blood pressure, the heart rate is 231. Accompanying the first rapid fall in blood pressure, the rate of heart beat decreases to 220. In the interval between this and the following convulsion, the heart rate falls to 212, which is somewhat more rapid than in the interval preceding the convulsion.

The relationship of blood pressure and heart rate to the muscular spasm remained constant through several records which were made. This relationship is also illustrated in figure 4. The initial rise in the blood pressure to 180 mm. is followed by the muscles becoming spastic; the limb is drawn up to the body, extended and drawn up, and extended a second time. This phase perhaps corresponds to the tonic phase of the convulsion, which was distinct in the severe convulsion described, but the tracing indicates a tetanic or tremulous element throughout. As the peak in blood pressure is reached (200 mm.) the muscles are observed to pass into a clonic spasm as indicated by the irregular wavy line. This spasm persists until the blood pressure has almost returned to the lower level marking the intervals between convulsions.

SUMMARY

Lesions were placed in the tuber cinereum of a series of dogs by injecting about 0.1 cc. of a weak solution (from 0.2 to 0.5 per cent) of mercuric chloride. In these animals, remaining otherwise normal in appearance, periodic convulsions developed.

The first convulsions, beginning from two to six hours after the operation, are usually of a mild form, characterized by disorientation, dilatation of the pupils, salivation, vasoconstriction, increase in rate of heart beat, and muscular spasms involving the face, jaws, and, to a varied extent, the anterior part of the body.

In the majority of cases, the convulsions continue gradually to become more frequent and more severe. The severe convulsions may

be described as follows: There suddenly occurs an appearance of fright and disorientation, accompanied by dilatation of the pupils, vasoconstriction and salivation. The appearance of these symptoms is followed instantly by rigidity, and the animal falls on its side. The rigidity soon gives place to clonic spasms involving the musculature of the entire body. Following the convulsion, the dog remains unconscious for several minutes, which is followed by a period of confusion and disorientation. The constant phenomena are tonic and clonic spasms, dilation of the pupil, salivation, marked increase in the rate of heart beat, a rise of 1 F. or more in body temperature, unconsciousness, frequent urination and occasional defecation. These convulsions usually last from one and a half to three minutes.

As the convulsions continue to occur with increasing frequency, the animal passes into status epilepticus. This phase usually lasts for two hours or more and results in death. It is characterized by a continuous state of coma, greatly increased rate of heart beat (from 230 to 260), dilatation of the pupils, salivation, a flaccid inactive state of the stomach and intestines and a temperature of from 108 to 111 F. Convulsions occur at intervals of a few seconds and are of from thirty to forty-five seconds in duration. The time from the beginning of the first convulsion to the death of the animal is usually from ten to twenty hours.

A few dogs exhibited periodic convulsions over a period of two or three days and made a complete recovery.

A study of the blood chemistry in these animals showed:

1. Neither the calcium or potassium content of the serums nor the potassium: calcium ratio were appreciably altered.

2. Nonprotein nitrogen seemed to increase with the increase in frequency and severity of the convulsions, but the results were not entirely constant.

3. Urea nitrogen followed the trend of nonprotein nitrogen but was even less constant.

4. Carbon dioxide combining power of the plasma decreased as the frequency and severity of the convulsions increased.

5. Invariably the sugar content of the blood rose steadily to about twice the normal value; then as the frequency and severity of the spasms increased, the blood sugar decreased until the animal died in the state of coma. Sugar values just before death were usually only one half of the normal value. Examination of the liver in three cases showed a total absence of glycogen.

It is believed that the lesion produced in the tuber cinereum is an irritative one causing a discharge of impulses from the nuclei in this region.

THE PERMEABILITY OF THE HEMATO-ENCEPHALIC BARRIER AS DETERMINED BY THE BROMIDE METHOD*

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CHICAGO

Between the blood serum and the cerebrospinal fluid is interposed a membrane or membranes consisting histologically of vascular endothelium in apposition with ependymal cells (choroid plexuses), or in apposition with pia-arachnoid (meninges). Stern and Gautier [1] have been pleased to call these membranes the hemato-encephalic barrier—for many purposes an excellent descriptive name, because in a measure it helps one to visualize some of the physiologic and clinical factors which involve these membranes. This partition acts in many ways, perhaps wholly, as a semipermeable membrane, permitting the passage of inorganic salts and other readily diffusable substances, such as dextrose and urea, into the spinal fluid, and excluding many blood serum constituents, notably colloids.

The arguments centering about the mechanism of the production of cerebrospinal fluid—whether or not it is formed by an active secretory process or is simply a dialysate—have been presented by numerous workers. The dialysate or physicochemical hypothesis, was formulated by Mestrezat in 1912. There are many factors disclosed by chemical analysis of the cerebrospinal fluid, and by the work of Dandy, Leonard Hill, Cushing, Weed and others on the physical and physiologic elements involved, which give considerable support to the Mestrezat theory. An able analysis and review of the physicochemical theory was made by Fremont-Smith.[2] However, sufficient information is not yet at hand to permit of dogmatic assertions concerning the entire mechanism of the production of cerebrospinal fluid. The histologic rôles played by the vascular endothelium, the ependymal cells, the choroid plexuses and the meninges proper have been emphasized by different investigators.

* Submitted for publication, Jan. 29, 1930.

* Read in part before the Philadelphia Psychiatric Society.

*From the Philadelphia Hospital for Mental Diseases, Byberry, Pa.

1. Stern and Gautier: Arch. internat. de physiol. **17**:391 (March) 1922; ibid. **20**:403 (March) 1923.

2. Fremont-Smith, Frank: The Nature of Cerebrospinal Fluid, Arch. Neurol. & Psychiat. **17**:317 (March) 1927.

Regardless of what attitude is held, certain facts are well recognized, namely, that certain constituents of the blood serum never appear in the spinal fluid under normal circumstances, and that inflammatory conditions involving these membranes frequently impair the integrity of this barrier.

A number of tests were devised seeking to determine meningeal permeability (the term meningeal here is taken to connote the entire barrier system and does not confine its meaning entirely to the meninges). Mestrezat devised the sodium nitrate test; Osborne [3] used sodium iodide; Kafka [4] presented the uranin test, and Weil and Kafka [5] the hemolysin test. Other methods may be based on the determination of the comparative concentrations of certain constituents of the blood serum and spinal fluid common to both, such as calcium, phosphates, chlorides, dextrose and urea. However, it appears that the most acceptable method used is one which utilizes some foreign substance, such as sodium nitrate or uranin. The latter methods have obvious drawbacks, in that in the nitrate test one is apt to measure increase and not decrease in the permeability, and the uranin test is purely a qualitative test.

In the selection of a suitable test substance, various factors appear to be of primary importance: (1) the test material, whether given by ingestion or intravenously, must reach a concentration in the blood serum sufficiently high and be maintained at that height for a sufficient period of time in order to insure its appearance in the spinal fluid; (2) the molecule or ion must be relatively small. Furthermore, in the case of substances that ionize, it has been demonstrated that negatively charged ions pass into the spinal fluid much more readily than do positively charged ones. Wittgenstein and Krebs [6] have demonstrated some of these factors in their work on dogs, using acid and basic dyes as well as colloidal dyes. They have shown the importance of the concentration factor as well as the importance of the ionization and electric charge.

In 1924, Walter [7] presented the bromide method for the determination of meningeal permeability. Bromide apparently meets all the requirements because it can be given in fairly large repeated doses, is eliminated from the body slowly, and therefore reaches a comparatively

3. Osborne, Earl D.: Iodin in the Cerebrospinal Fluid, J. A. M. A. **76**:1384 (May 21) 1921.

4. Kafka: Untersuchungen zur Frage der Entstehung, Zirkulation und Funktion der Cerebrospinalflüssigkeit, Ztschr. f. d. ges. Neurol. u. Psychiat. **13**:192, 1912; ibid. **15**:482, 1913.

5. Weil and Kafka: Ueber die Durchlässigkeit der Meningen, etc., Wien. klin. Wchnschr. **24**:335, 1911.

6. Wittgenstein and Krebs: Ztschr. f. d. ges. exper. Med. **49**:553, 1926.

7. Walter, F. K.: Arch. f. Psychiat. **79**:363, 1927; Monatschr. f. Psychiat. u. Neurol. **60**:283, 1926; Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:522, 1925; **97**:192, 1925; **99**:548, 1925; Klin. Wchnschr. **6**:1618, 1927.

high concentration in the blood stream; it has also a negative charge when ionized. It slowly replaces some of the chlorides in the blood and is retained over a sufficient period of time for appreciable quantities to appear in the spinal fluid.

Method.—The patient is given 0.03 Gm. of sodium bromide per pound of body weight daily for five consecutive days. On the morning of the sixth day, samples of the blood and spinal fluid are collected. The blood serum is used and diluted and the protein is precipitated. The spinal fluid is deproteinized in a similar manner and both solutions are filtered. The respective filtrates are treated with gold chloride solution which reacts with the bromide, giving from a yellowish to a golden brown color (Guareschi test for bromide). The relative concentrations are determined by comparison in a colorimeter.

In his early work Walter used nitric acid as a protein precipitant. Together with others who utilized the Walter method, Hauptmann[8] found nitric acid unsatisfactory and proposed and used trichloracetic acid. In our work we found that occasionally 20 per cent trichloracetic acid apparently did not completely precipitate spinal fluid protein, and that the spinal fluid filtrates contained sufficient unprecipitated protein to interfere with the gold chloride reagent. We then utilized nascent tungstic acid for deproteinization with the ordinary reagents used in the Folin-Wu system of blood analysis.

Technic.—The blood is collected in a 15 cc. pyrex centrifuge tube, allowed to clot and centrifugalized; the clear serum is used. Three cubic centimeters of serum is pipetted into a small Ehrlenmayer flask, 8 cc. of distilled water added, then 2 cc. of 10 per cent sodium tungstate and then 2 cc. of two-thirds normal sulphuric acid, drop by drop, whirling the flask after the addition of each few drops. A tightly fitting stopper is applied, the mixture is shaken vigorously and allowed to stand for ten minutes and then filtered through a heavy grade of filter paper (Whatman no. 2).

To 6 cc. of spinal fluid, introduced into a similar flask, 2 cc. of distilled water is added and then 1 cc. of the sodium tungstate solution and 1 cc. of the two-thirds normal sulphuric acid in the same manner; this is shaken, allowed to stand ten minutes and filtered. The dilution of blood is now three times that of the spinal fluid. As it has been found that the concentration of bromide in the serum is usually three times that of the spinal fluid, they will be more nearly comparable when the color is developed.

Two cubic centimeters of the clear blood filtrate is pipetted into a small test tube and 0.2 cc. of a 0.5 per cent gold chloride solution added, with the development of the color already noted. The same quantity of spinal fluid filtrate is treated in the same manner. The respective blood and spinal fluid colors are then compared in a colorimeter. If the respective shades of color diverge widely they are compared with known standards of gold bromide solution.

It is not necessary to obtain absolute values, as one is interested only in the ratio of distribution of bromide between the serum and spinal fluid. This ratio is known as the permeability quotient. It has been found normally that the blood serum contains about three times the concentration of bromide that obtains in the

8. Hauptmann: Untersuchungen ueber die Blut-Liquorpassage, Ztschr. f. d. ges. Neurol. u. Psychiat. **100**:332, 1926; Untersuchungen zur Pathogenese alkoholischer Psychosen, Zentralbl. f. d. ges. Neurol. u. Psychiat. **44**:499, 1926; Klin. Wchnschr. **4**:1629, 1925; Ztschr. f. Nervenh. **100**:91, 1927.

spinal fluid. In other words, the normal permeability quotient averages about 3. This figure is obtained by dividing the concentration in the blood by the concentration in the spinal fluid. A high permeability quotient, therefore, means diminished permeability and a low quotient increased permeability.

What is to be considered a normal range of permeability quotients is not entirely settled, and Walter himself has had occasion to change his range of normal quotients. He first held that the normal range fell between 2.9 and 3.3, and later changed the latter figure to 3.5. These figures are based on his own work and that of Sunderhauf,[9] who utilized psychoneurotic types and patients with somatic diseases that were apparently not related to conditions of the nervous system as normal controls. Malamud[10] took the figures from 2.8 to 3.2 arbitrarily as the normal range of permeability quotient. In presenting our results we have charted the figures and have used the permeability quotient of 3 merely as a line of division for comparative purposes, and have not given any percentage results based on a so-called normal range.

COMPARISON OF OUR RESULTS WITH THOSE OF OTHER WORKERS

In all, 183 patients in the Philadelphia Hospital for Mental Diseases were studied by Walter's bromide method; they included 108 men and

TABLE 1.—*Range of Permeability Quotient in Thirty-Two Cases of Dementia Paralytica*

	Permeability Quotient							
	1.5 to 2.0	2.0 to 2.2	2.2 to 2.5	2.5 to 2.8	2.8 to 3.0	3.0 to 3.2	3.2 to 3.5	Total
Number of cases	8	2	10	10	2	0	0	32

75 women. We have not reduced our figures to a percentage basis because this would require an arbitrary assumption of normal ranges. The figures as arranged indicate tendencies and can be used for comparison, one group with another and with the results of other workers.

Dementia Paralytica (table 1).—In no instance was a permeability quotient above 3 encountered. Our observations are in distinct accord with those of all other workers, namely, that the majority of patients with dementia paralytica show increased permeability. If Malamud's range of normal (2.8 to 3.2) is applied to this series, only two cases could be called normal and the other thirty show increased permeability. This number included patients with dementia paralytica unsuccessfully treated with malaria before admission to our hospital.

Dementia Praecox (table 2).—In 83 cases we found that the distribution of cases was equally divided on either side of the figure 3, and that over one-half the cases showed a normal permeability quotient

9. Sunderhauf: Ztschr. f. d. ges. exper. Med. **55**:378, 1927.

10. Malamud, William; Fuchs, Douglas M., and Malamud, Nathan: Barrier Between the Blood and the Cerebral Spinal Fluid, Arch. Neurol. & Psychiat. **20**:780 (Oct.) 1928.

(using Malamud's normal range). If Walter's [7] normal range is accepted (from 2.8 to 3.5), about 69 per cent showed normal permeability, 24 per cent increased permeability and only 7 per cent diminished permeability. In this group our observations are not in accord with those of Walter, Hauptmann, Störring [11] or Malamud, each of whom held that the majority of schizophrenic patients showed diminished meningeal permeability. In fact, Hauptmann thought that the bromide test might be used as a differential diagnostic criterion between schizophrenia and cyclothymia. Our observations are more in agreement with

TABLE 2.—*Range in Permeability Quotient in Eighty-Three Cases of Dementia Praecox*

	Permeability Quotient							
	2.1 to 2.3	2.3 to 2.5	2.5 to 2.8	2.8 to 3.0	3.0 to 3.2	3.2 to 3.5	3.5 to 3.9	Total
Catatonic	1	3	9	14	12	8	6	53
Hebephrenic	..	..	4	6	4	4	..	18
Paranoid	..	..	..	1	2	..	..	3
Simple	..	1	2	1	3	..	..	7
Untyped	..	..	..	..	2	..	..	2
Number of cases	1	4	15	22	23	12	6	83

TABLE 3.—*Range of Permeability Quotient in Twenty-Six Cases of Manic-Depressive Psychosis*

	Permeability Quotient							
	2.1 to 2.3	2.3 to 2.5	2.5 to 2.8	2.8 to 3.0	3.0 to 3.2	3.2 to 3.5	3.5 to 3.8	Total
Manic	0	0	8	0	0	0	0	8
Depressed	..	2	3	6	1	..	1	13
Mixed	2	..	..	2	..	..	..	4
Cyclic-schizoid	..	..	..	..	..	1	..	1
Number of cases	2	2	11	8	1	1	1	26

Jacobi and Kolle,[12] Metzger and Hoffmann,[13] Kral [14] and Strecker.[15] The catatonic patients showed the widest variation in both high and low quotients and the paranoid patients the least variation. However, in the entire group of 183 cases, all patients with one exception (a depressed phase of manic-depressive psychosis) showing a permeability quotient of 3.5 or over, totaling six in number were schizophrenic

11. Störring: Arch. f. Psychiat. **86**:567, 1929.

12. Jacobi and Kolle: Bemerkungen zu der Arbeit von Hauptmann, Monatschr. f. Psychiat. u. Neurol. **60**:265, 1926.

13. Metzger and Hoffmann: Ztschr. f. d. ges. Neurol. u. Psychiat. **107**:618, 1927.

14. Kral: Zur Pruefung der meningealen Permeabilitaet mittels der Walterschen Brommethode, Ztschr. f. d. ges. Neurol. u. Psychiat. **115**:158, 1928.

15. Strecker: J. Ment. Sc. **74**: 73 (Jan.) 1928.

patients. It appears that markedly diminished permeability is found in a definite number of schizophrenic patients, but not necessarily in the majority.

Manic-Depressive Psychosis (table 3).—According to our studies there appears to be a tendency toward increased permeability in this

TABLE 4.—*Range of Permeability Quotient in Fourteen Cases of Alcoholic Psychosis*

	Permeability Quotient						
	2.3 to 2.5	2.5 to 2.8	2.8 to 3.0	3.0 to 3.2	3.2 to 3.5	3.5 to 3.9	Total
Deterioration	..	2	..	..	..	..	2
Acute hallucinosis	1	2	2	..	..	..	5
Depression	..	1	..	..	..	..	1
Confusion	..	1	2	..	..	..	3
Paranoid	..	..	2	..	..	..	2
Without psychosis	..	..	..	1	..	..	1
Number of cases	1	6	6	1	0	0	14

TABLE 5.—*Range of Permeability Quotient in Sixteen Cases of Postencephalitis*

	Permeability Quotient							
	1.5 to 2.0	2.0 to 2.2	2.2 to 2.5	2.5 to 2.8	2.8 to 3.0	3.0 to 3.2	3.2+	Total
Number of cases	..	1	3	3	6	2	1	16

TABLE 6.—*Range of Permeability Quotient in Four Cases of Senile Psychosis*

	Permeability Quotient					
	2.3 to 2.5	2.5 to 2.8	2.8 to 3.0	3.0 to 3.2	3.2 to 3.5	Total
Number of cases	0	1	2	1	0	4

TABLE 7.—*Range of Permeability Quotient in Eight Cases of Psychosis with Cerebral Arteriosclerosis*

	Permeability Quotient					
	2.3 to 2.5	2.5 to 2.8	2.8 to 3.0	3.0 to 3.2	3.2 to 3.5	Total
Number of cases	1	3	1	3	0	8

group, but not sufficiently marked to warrant emphasis. About one half of the cases were below 2.8, the lower limit of normal permeability quotient, according to Walter.

Alcoholic Psychosis (table 4.)—The distribution of quotients in this group shows a resemblance to table 3 of the manic-depressive group. There was no access to the patients with acute alcoholic psychosis, such as those with delirium tremens or dipsomania, to compare them with

patients with chronic manifestations of alcoholic psychosis (such as constituted our material). According to Walter and Hauptmann, these acute types are associated with increased permeability. From our observations it would appear that a certain proportion of the chronic forms of alcoholic psychosis show a similar tendency.

Postencephalitis (table 5).—These cases were all of the chronic type. No deductions can be drawn that would be of any particular significance.

Senile Psychosis (table 6) *and Psychosis with Cerebral Arteriosclerosis* (table 7).—Patients with these conditions showed nothing of marked note with the possible exception of the fact that the arteriosclerotic patients seemed to show some tendency toward increased permeability. Malamud in an examination in sixteen cases of psychosis with cerebral arteriosclerosis found that there was a marked tendency toward increased permeability. Our observations in a measure tend to substantiate his conclusions.

SUMMARY

A series of 183 cases of mental disorder studied for the determination of meningeal permeability by the Walter bromide method is presented.

In dementia paralytica the majority of cases showed a marked tendency toward increased meningeal permeability.

In dementia praecox the majority of patients presented normal permeability (about 69 per cent), about 24 per cent increased permeability and the remainder diminished permeability.

In the cases of chronic alcoholic psychosis there appears to be a tendency toward increased permeability. A similar tendency is shown to some degree in psychosis with cerebral arteriosclerosis.

About one-half the cases of manic-depressive psychosis showed normal and the other half increased permeability.

In the postencephalitic cases and in senile psychoses no particular tendencies were shown.

The Walter bromide method does not appear to afford distinct aid in the differential diagnosis of mental disease, especially as between dementia praecox and manic-depressive psychosis.

The wide range of permeability quotients, such as the ones displayed in the catatonic cases of dementia praecox, may be evidence that one is dealing with schizophrenias as a plural concept in contrast with schizophrenia as a unity.

It may be that the marked deviation of the permeability quotient from the normal range occurring in other psychoses may have a discoverable organic basis such as occurs in dementia paralytica.

DISCUSSION

DR. JOSEPH C. YASKIN: A striking observation in these cases is the marked increase in the permeability of the hemato-encephalic barrier to sodium bromide in dementia paralytica. When one considers that in this disease there is a marked lymphocytic infiltration throughout and that the permeability to arsenic compounds, mercury, etc., is markedly decreased, one is surprised at the observations recorded. It is to overcome this impermeability to antisyphilitic remedies that Gilpin and his co-workers have instituted the combined spinal drainage and intravenous medications. It would be interesting to know the explanation of the increased permeability to sodium bromide.

DR. C. A. PATTEN: What are the permeability quotients in normal persons? Does bromide in any way injure the barrier, thereby in itself affecting the results?

DR. CHAS. W. BURR: Are not most of the cases reported around 3?

DR. YASKIN: Is it not surprising that in dementia paralytica, in which one knows that the meninges are frequently thickened, there is increased permeability? This matter is of importance because the frequent failure of the arsenic compounds to benefit patients with neurosyphilis is alleged to be due to the failure of the arsenic to penetrate to the nerve tissue.

DR. W. L. LONG: The ratio of the concentration of the chloride in the plasma to that in the corpuscle is rather constant under ordinary conditions. If the blood is made acid by the pouring in of lactic acid from one minute of violent exercise, practically all of the chloride leaves the plasma and goes into the corpuscles. The concentration of chloride in the urine then falls practically to zero. As the bromide is so closely related to the chloride ion, the conditions under which the bromide ion concentration is measured, in both the blood and the spinal fluid, should be constant. Exercise, diet, time of day, etc., probably cause variations in the ratio of concentration of the bromide ion in the blood to its concentration across the hemato-encephalic barrier.

DR. GORDY: We had no normal controls. We accepted the so-called normal figures of Walter and those of Sunderhauff, who based their figures on permeability quotients, results in psychoneurotic patients and in patients with somatic diseases having no apparent relation to the central nervous system. Whether these patients might be called normal is open to question. It is hard to get a normal person. In the matter of any possible toxic action by sodium bromide on the barrier, I can say only that I know of no pharmocologic action of bromide which would lead one to suspect toxic damage of such a character. This action from a physical point of view is probably similar to that of any salt in dilute solution.

In answering Dr. Burr, I should like to say that most of our cases fall around the figure 3; we have used this as a line so that the cases would show the degree of deviation from 3; the charts thus can be utilized for comparative purposes, and we have accepted the figure 3 tentatively as an arbitary normal.

In answering Dr. Yaskin, the permeability as demonstrated for bromide does not necessarily hold for the molecules of arsenic compounds used in therapy. In discussing the matter of permeability in relation to thickened membranes as a result of acute or chronic inflammatory processes, one must not confuse physical thickness with what might be termed barrier integrity. For example, in the process of an acute inflammation, one knows that swelling and plastic exudate contribute to physical thickness, but at the same time the toxic agent causing the inflammation permits albumins and other colloids, which are normally not found outside of the blood vessels, to get out into tissue spaces—another type of increased permeability, but referring to the permeability of vascular endothelium.

LESIONS OF THE FRONTAL LOBE

A REVIEW OF FORTY-FIVE CASES *

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Fifteen years ago, general discussions on tumors of the brain were usual; today no such general discussion is of value. With the large series of cases of tumors of the brain that have been collected in neurosurgical clinics, a different type of study must be employed. To arrive at any satisfactory conclusions, one must consider special groups, either certain pathologic groups or lesions located in certain regions of the brain. In this article I shall consider the lesions of the frontal lobe and review my series of cases. Two years ago, I discussed this subject at a joint meeting, in London, of the British and American Neurological Societies. At that time I had twenty-five verified cases. During the past year and a half twenty more cases have been added. In these forty-five cases, all but one of the patients were operated on, with a mortality of 17 per cent.

The diagnosis of a lesion of the frontal lobe still remains a fascinating problem, and the differential diagnosis often requires the most careful analysis of the observations to enable one to come to a decision. My purpose in that study was to determine which symptoms or signs had been found most dependable in arriving at a diagnosis. The present paper is a check-up on those observations.

While, in general, observations on the entire group have confirmed the views based on the first twenty-five cases, I have been obliged to modify my views somewhat. At that time I pointed out that the two most dependable symptoms were facial weakness of the central type affecting emotional movements on the side opposite the lesion, and a mental change. Both these symptoms have still proved of great value, but they have not been noted in so large a number of cases; however, the variation has been only around 10 per cent, so that I still feel confident that these two symptoms are of the greatest help.

In studying these cases, however, there are other points that have stood out more prominently than was at first apparent, and they are of great interest: first, the frequent occurrence of convulsions, and second, the observation of nystagmus.

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* Read at a meeting of the Chicago Neurological Society, Jan. 16, 1930.

These four symptoms—facial paresis, mental change, convulsions and nystagmus—I shall consider first.

Facial paresis is common because the facial center in the precentral gyrus lies farther forward than any other; consequently, it is the first one pressed on. Why the facial weakness should be more often noted on emotional innervation of the face is not so readily explained. There is an anatomic observation that I made a good many years ago in some work on the optic thalamus which may explain this. In degeneration experiments, I found a tract of fibers passing from the cortical face center to the thalamus. There was no such tract from either the arm or the leg center. When the thalamus is not functioning normally, patients show peculiar emotional disturbances; these were described years ago by Roussy and Kinnier Wilson. With increased intracranial pressure, this corticothalamic pathway may be thrown out of function, and what might be spoken of as a thalamic type of facial paresis develops. The facial paresis is peculiar in that repeatedly it has happened that the patient or a relative has insisted that the patient always has had this facial asymmetry; yet after removal of the tumor, it has disappeared. In one striking instance, the young man's fiancée said that she had frequently commented on his laughing only on one side of the face; after removal of the tumor, the innervation of both sides of the face was the same when the patient laughed.

The mental changes that patients with a lesion of the frontal lobe show are extremely varied and difficult to describe, and still more difficult to classify into special groups. There have been some patients in this series who have had religious hallucinations and have been in an excited state; others have been depressed and monosyllabic, but this is not the mental change that is particularly characteristic. The patient's original temperament may influence this element of the mental change. Thus a patient who has always been jovial and happy may show a certain reaction, while the morose, quiet type may show a different reaction. The change may be an exaggeration of the patient's normal state or just the opposite of his normal state. Why this should be, I must leave to psychologists or psychiatrists to explain, but I have wondered whether exaggeration of a patient's normal state may be in the nature of an irritative phenomenon, while the occurrence of a condition opposite to the patient's normal state may be an inhibition, by destruction or pressure, of the normal reactions.

The features that are constant in the mental change in cases of lesions of the frontal lobe are two: (1) Loss of memory for recent events, with good memory for events that occurred six months or more before. Thus, a patient who had made a thirty-six hour trip from Texas, the same day remembered nothing about it or where he was; yet he could

Summary of Forty-Five Cases of Lesions of the Frontal Lobe

Number of cases of lesions of frontal lobe		45
Number of patients operated on	44	
Number of deaths (one of these not operated on)	9	

TYPE OF LESION

Glioma	18	Chronic inflammation and foreign body	1
Endothelioma	14	Basal carcinoma	1
Gumma	2	Degeneration of frontal lobe	1
Abscess	5	Bone cyst	1
Arachnoidal cyst in region of sylvian fissure	1	Tuberculoma	1

SIDE OF LESION

Left side	27
Right side	18

HEADACHE AND VOMITING

Headache	35
Vomiting	22

RIGHT OR LEFT HANDED

Right handed patients	44
Left handed patients	1

OPHTHALMOSCOPIC OBSERVATIONS

Normal eyegrounds		7
Slight blurring		6
Distinct difference between the two sides		7
On side of lesion	4	
On side opposite lesion	3	
Choked disk, bilateral		26
Unilateral atrophy		1

VISUAL FIELDS

Normal fields	14
Partial homonymous hemianopsia due to encroachment of tumor on adjacent temporal lobe	5
General contraction due to high grade of choking	6
Central scotoma	2
Could not be taken because of lack of cooperation of patient	19

CONVULSIVE MOVEMENTS

Focal convulsions	11
General convulsions	9

NYSTAGMUS

Nystagmus, lateral	8

MOTOR CHANGES

Hemiparesis, varying from slight involvement to almost complete hemiplegia	16
Facial weakness on side opposite lesion	32
Facial weakness only, with no other motor paresis	15
Weakness of arm or leg without facial involvement	14

REFLEXES, UPPER AND LOWER EXTREMITIES

Normal reflexes	16
Pathologic reflexes:	
Oppenheim and Babinski, but no other pathologic reflexes	17
Ankle or patellar clonus in addition to Oppenheim and Babinski signs	10

REFLEXES, ABDOMINAL

Normal reflexes	19
Not obtained on either side due to fat abdomen	11
Diminished on side opposite lesion	9
Diminished on side of lesion	5

SENSE OF SMELL

Subjective disturbance of smell	3
Loss of smell, both sides	5
Loss of smell on side of lesion	5
Sense of smell normal	33
Could not be tested	2

UNILATERAL TREMOR

On side opposite lesion	3
On side of lesion	2

ADIADOKOKINESIS

Adiadokokinesis on side opposite lesion	1

SPEECH DISTURBANCES

No speech disturbances			25
Speech disturbances			20
Verbal aphasia, marked	5	Nominal aphasia, with slurring and clumsiness in word expression	1
Verbal aphasia, slight	7	Hesitancy and slowness in speech	4
Nominal aphasia	3		

MENTAL CHANGES (Including Emotional Changes)

No mental changes			12
Gumma	1	Bone cyst	1
Abscess in a child	4	Astrocytoma	2
Endothelioma	4		
Mental changes			32
Emotional changes	17	Emotional without mental change	4
Impulsive laughter or crying	9	Both mental and emotional change	13
Morose or irritable	7		
Indifferent	1		
Mental changes without emotional changes	19		

X-RAY OBSERVATIONS

Roentgenograms showed shadows interpreted as tumor shadows	13
Roentgenograms showed pressure skull	14
Roentgenograms showed foreign body	1
Roentgenograms showed no changes	17

INJECTION OF AIR

Injection of air was undertaken in 13 cases, but in only 9 was air put in; in 6 injection of air established the diagnosis but in 5 of these it merely confirmed the diagnosis; all 9 showed collapsed ventricles. The condition in one case was first diagnosed as cerebellar tumor; this patient would have been spared the cerebellar operations had the injection of air been done first

tell the number of the house and street where he had lived ten years before. Another patient remembered historic facts that he had lived through but could not remember the name of the President of the United States. Occasionally, this loss of memory manifests itself in a most unusual way. One patient had been a gifted instructor in architecture at a university. He lost his position because he could no longer draw. He told me afterward that he could no longer draw a straight line, and while he had been able to sketch well, all this faculty disappeared and then returned after removal of his tumor. (2) A total lack of concern about the disease or its outcome. Thus a patient when told he would have to have his head operated on, laughed about it and was perfectly unconcerned. Euphoria is a frequent feature in these cases.

Some patients have emotional changes, weeping or laughing, but these are independent of the other mental changes; that is, a patient may have only an emotional instability, but no other mental change, or an emotional instability and other mental changes may be present in one and the same patient. The emotional changes, however, have been much less common than the memory defects, but it has occurred only four times that a patient had an emotional disturbance without some other mental change. I succeeded in photographing in an attack an excellent example of emotionalism associated with a tumor of the frontal lobe. This man, while he was crying, had apparently no sensation of grief and none of the sensations that one might expect to be associated with his appearance.

As my series of cases of lesions of the frontal lobe has increased, I am more and more impressed with the frequency with which patients have convulsions. I still believe that convulsions are an irritative phenomenon arising, as a rule, from the cortex. Certainly, this seems to be true of focal convulsions as well as of general convulsions. General convulsions, it seems, are peculiarly characteristic of disturbances in the frontal lobe, but why the frontal lobe should give rise to general convulsions is not clear. There is no physiologic basis on which to explain this, and one would be obliged to speculate as to the possible cause without having any sound basis to go on. At present one must be content merely to record the fact.

The fourth symptom which, though not frequent, is an interesting one, is nystagmus. Its chief interest lies in the fact that it tends to confuse the diagnosis. Some years ago, the German literature contained numerous reports of cases of lesions of the frontal lobe in which the condition was mistaken for cerebellar tumors, and no doubt the presence of nystagmus was the principal cause for this mistake. How is this symptom explained? There are but two possible ways that I can see. There is a center in the frontal lobe, in front of the face center, which

causes conjugate movements of the eyes. Intermittent stimulation of this center might produce a nystagmoid-like movement. It is significant that the movements of the eyes in a case of lesion of the frontal lobe are irregular twitchings, not so regular as in cerebellar nystagmus. The other explanation is that this is an evidence of von Monakow's theory of diaschisis. I confess I have never been enthusiastic about that theory or impressed with its value. If one accepts that theory, one must give up much of the theory about cerebral localization. This I am unwilling to do, and I can see no reason for discarding it. In one case of this series, I made the mistake and twice explored a cerebellum on account of nystagmus and ataxia and only then realized my error and finally removed a meningioma from the right frontal lobe. In this case I should have realized my mistake at the first operation when I had difficulty in striking the ventricle, as it was not dilated. Since that experience of a good many years ago, I believe I have learned to distinguish between a true cerebellar nystagmus and the nystagmoid movements that occur with lesions of the frontal lobe. In one of these, however, injection of air had to be used to establish the diagnosis. In a doubtful case, when the diagnosis lies between a frontal and a cerebellar lesion, a ventricular estimation or ventriculogram at once clears up that point.

One of the questions that has interested me greatly, and puzzled me even more, is the difference in function between the right and the left frontal lobes. Aside from the speech disturbance, which was present to some extent in almost all the cases of lesion of the left frontal lobe, what difference can one detect in the mental changes produced by the right or the left frontal lobes? Reviewing the cases with this point in mind, I have found that there has not been a single mental change with a lesion on one side, that could not also be observed when the lesion was on the other side. Both frontal lobes seem to be equally concerned with memory, and, when impaired, memory seems to be affected in the same way. The decision whether a mental disturbance is due to a psychosis or to an organic frontal lesion is at times staggeringly difficult, and I have no hesitation in saying that to pass on that point I always call in a psychiatrist. I have observed that even the psychiatrist, however, is often at a loss to determine which he is dealing with. The only way to decide this point, it has seemed to me, is to weigh the other evidence and, if the other symptoms indicate an organic lesion, to assume that the mental phenomena are due to the same cause. The general principle of trying to explain the various cerebral symptoms in a case by a single pathologic process, rather than by several lesions, is, I think, a sound one.

One of the group of symptoms that has been described as characteristic of lesions of the frontal lobe is that of Foster Kennedy—optic atrophy on the side of the lesion and choked disk on the opposite side.

This, or something approaching it, was observed only once in the forty-five cases, and in that instance there was unilateral optic atrophy, but a normal disk on the opposite side. That this was such a rare observation can be explained only by the fact that the type of lesion that produces these symptoms is one situated on the under surface of the frontal lobe where it exerts direct pressure on the optic nerve of the same side. This is a rare location for a lesion and, as a rule, as in my own case, is associated with unilateral exophthalmos. The lesions in these cases are, as a rule, meningiomas growing from the olfactory groove and may actually invade the orbit. This clinical picture, however, may be produced by any lesion of the frontal lobe which exerts pressure on the base. There is one case in the series which is of particular interest, as it throws light on the development of this clinical picture and also brings up an important point about which I, for one, had been much puzzled, namely, the length of time a patient can be blind in one eye and yet regain his vision. The case is that of a boy who had an abscess of the right frontal lobe; fully four weeks before admission, he was totally blind in the right eye. After opening the abscess, which was under great pressure, vision in the right eye returned with remarkable rapidity. He was able to recognize objects with the right eye twenty-four hours after the abscess was opened.

Prior to this experience I had felt that if vision is gone for but a few hours the prospect of recovery is poor. Everyone has seen exceptions to this in cases of pituitary tumor, but in those cases there is no associated choked disk, while here there was a choked disk. In this case, then, there was a physiologic block of a sensory cranial nerve which was present for at least four weeks and after release of the pressure, the function returned. This fact makes one a bit more optimistic about the possibility of the patients in cases of tumor getting back some vision if it has not been blotted out too long. I know of no satisfactory explanation why one nerve withstands pressure much longer than another.

SUMMARY

The points that stand out in this study of a group of lesions of the frontal lobe, and are the most dependable signs of a lesion are:

1. The mental change. This is characterized particularly by a loss of memory for recent events and indifference as to the illness and to the surroundings. At times, this mental change resembles a euphoric state: at others there may be marked depression.

2. A facial weakness on the side opposite the lesion, sometimes associated with involvement of the pyramidal tract of the opposite side, but often the first evidence of involvement of the pyramidal tract.

In addition to these signs, there may be disturbances of speech, occasionally defects in the visual field when the temporal lobe has been encroached on, and some of the rare symptoms that occur so occasionally that they cannot be considered diagnostic. When the first two symptoms alone are present, without changes in the eyegrounds, it may be necessary to resort to injection of air. In a number of cases this has cleared up uncertainty, and when there is a possibility of confusion between a posterior fossa and a frontal lesion, the study of the ventricles is essential.

DISCUSSION

DR. GEORGE W. HALL: Dr. Sachs has brought out the diagnostic points extremely well, but there are two things about which I should like to inquire. First, as to his experience concerning tumors of the olfactory groove; I reported an interesting case of tumor of the olfactory groove in which the optic nerve on the opposite side remained normal throughout. I reported this case about two years ago. The usual observations were that the nerve on the opposite side from the tumor showed evidences of papilledema. Another thing is that while I have not seen so many cases of tumors of the frontal lobe as has Dr. Sachs, the convulsions in those cases which I have seen have not occurred early but rather in the advanced stages. Another point is in the tabulation concerning the loss of smell; those cases in which loss of smell was recorded were they not endotheliomas rather than other forms of tumor? The statement made concerning the error of mistaking a tumor of the frontal lobe for a tumor of the cerebellum, I think should receive special attention. It is not so unusual after all, that tumors in which the symptoms point toward involvement of the frontal lobe, clinically speaking, are shown by employing ventriculography, to be basal tumors instead.

DR. PETER BASSOE: I wish to ask about the symptom of "forced groping and grasping" that has been described by some English observers as occurring in cases of tumor of the frontal lobe. Did Dr. Sachs observe it?

DR. PERCIVAL BAILEY: I think that Dr. Sachs' presentation is good evidence that knowledge of tumors of the brain is becoming more or less standardized; probably the explanation for that is not only the extent of the experience of different men who are working with tumors, but also the close association of the men who are doing this work and who are members of the Neurosurgical Association, which meets every six months and discusses these subjects.

The method of analysis and the method of determining the frequency of certain symptoms in tumors of the frontal lobe which Dr. Sachs has employed, is useful. In many of the neurologic treatises, one might be led to believe that anosmia is as frequent as any other symptom.

Another point that should be emphasized is that the symptoms depend not only on the localization of the lesion but also on its pathologic nature; I think that we are beginning to think more and more pathologically in dealing with tumors of the brain, although that attitude is not as widespread as it should be. Recently, I received a questionnaire concerning tumors of the hypophysis. Just as in tumors of the frontal lobe, we have passed the point at which we can talk about tumors of the hypophysis as an entity. Two types commonly occur, one a cystic tumor, the other an adenoma of the hypophysis; one does not get far if one attempts to discuss these tumors as one. If you put in different columns the symptomatology and treatment, the result is entirely different for each type. To discuss them as one type of tumor leads to confused conclusions. In tumors of the frontal lobe such an analysis as Dr. Sachs has made is essential, but one must consider them from a pathologic standpoint as well and strive to make a preoperative pathologic diagnosis.

DR. G. B. HASSIN: I wish to inquire how often Dr. Sachs has noted the phenomenon of cerebellar ataxia in tumors of the frontal lobe.

DR. MEYER SOLOMON: Has Dr. Sachs found any cases of complete palsy of the third nerve in basal cell frontal tumors?

DR. SACHS: I cannot answer whether loss of smell was associated with any particular type of tumor, for I did not review the cases from that point of view. To my mind, the series is entirely too small to draw any conclusions of that sort; that is, to claim that a certain symptom occurs more frequently with a certain type of pathologic lesion than with another. I think that Dr. Bailey's point is an excellent one, but in order to express any intelligent opinion, one would have to have a far larger group of cases than this. In my series there were about as many gliomas as there were meningiomas. Anosmia occurred too rarely to be of any great value in diagnosis anyway.

My only reason for mentioning unilateral tremor was to point out how very rarely it occurred and therefore of how little value this symptom is. It occurred as frequently on one side as on the other.

I am not familiar with the forced grasping reflex as being particularly characteristic of tumors of the frontal lobe. I have seen it only on one or two occasions, and then it occurred with a subcortical tumor in the precentral gyrus. Ataxia occurred but once in the entire series.

Dr. Bailey's other point I think is also of great importance, and that in my experience has been the serious drawback in textbooks on neurology. Symptoms are mentioned as occurring in certain lesions, but their frequency is not brought out.

I have seen one case of tumor growing from the edge of the greater wing of the sphenoid that produced a paralysis of the third nerve, but this I considered more a tumor of the middle fossa than of the frontal lobe. Aneurysm of the internal carotid just before it joins the circle of Willis is the most common cause of paralysis of the third nerve and this, of course, is also a lesion of the middle fossa.

THE TAKATA-ARA COLLOIDAL TEST WITH SPINAL FLUID*

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The neurologist and clinical pathologist are engaged in a search for simple, yet reliable, tests for pathologic phenomena in the spinal fluid. To the rather imposing array of colloidal tests applied in recent years has been added the Takata-Ara test, developed by two Japanese investigators, Maki Takata and Kiyoshi Ara in 1925. The practical value of this test, which is otherwise referred to as the sublimate-fuchsin reaction, has been studied by a number of workers in various European clinics. Blum,[1] who tested the fluid from 200 patients, claimed that it is a valuable means of differentiating syphilitic from metasyphilitic nervous or mental disease. He found positive reactions in 98 per cent of cases of dementia paralytica and tabes. He pointed out that a serious drawback is the sensitiveness of this test to slight admixtures of blood.

Other workers are equally emphatic in stressing the nonspecificity of this reaction. Rohrs and Kohl-Egger[2] found that fluid in many organic brain diseases, nonsyphilitic in origin, shows a relatively heavy precipitate of the type found in dementia paralytica. Moreover, a mixed type of reaction often appears, the red coloring of meningitis and the precipitation of the metasyphilitic reaction occurring in the same tube, so that a distinction no longer obtains and, therefore, a differential diagnosis becomes uncertain.

In view of these radical differences of opinion as to the reliability of this test as an adjunct in diagnosis, spinal fluids from patients with both syphilitic and nonsyphilitic mental and nervous disturbances were studied in this series, and the gum mastic colloidal reaction in all instances was used as a basis for comparison. The latter colloidal reaction has been generally accepted as a reliable and accurate test, easy to perform and simple to interpret and on a par with the colloidal gold test, as has been well demonstrated by Keidel and Moore[3] and by

* Submitted for publication, April 22, 1930.

1. Blum, E.: Takata-Ara's Colloidal Chemical Reaction as a Valuable Means of Differentiating Syphilitic from Metasyphilitic Nervous Diseases, Ztschr. f. d. ges. Neurol. u. Psychiat. **110**:504, 1927.

2. Rohrs, E., and Kohl-Egger: Nonspecificity of Takata-Ara Reaction, Deutsche Ztschr. f. Nervenh. **101**:1, 1928.

3. Keidel, A., and Moore, V. E.: Comparative Results of Colloidal Mastic and Colloidal Gold Tests, Arch. Neurol. & Psychiat. **6**:183 (Aug.) 1921.

Cockrill.[4] Parallel readings were taken on 180 spinal fluids from patients admitted to the psychopathic ward of Cleveland City Hospital, including not only patients with dementia paralytica and tabes but also those with organic conditions such as cerebral arteriosclerosis, embolism, brain tumor and meningitis. In addition, fluids from persons with various clinical forms of alcoholism and the major types of primary psychoses were tested with the gum mastic and the Takata-Ara reaction.

The technic followed in this series is that described by Blum and is as follows:

To 1 cc. of spinal fluid one drop of 10 per cent sodium carbonate solution is added; after shaking this is mixed with 0.3 cc. of equal parts of a 0.5 per cent solution of corrosive sublimate and 0.02 per cent fuchsin solution. With normal

Results of the Takata-Ara Colloidal Test with Spinal Fluid

Type of Psychosis	Number of Cases	Positive Gum Mastic and Positive Takata-Ara	Negative Gum Mastic and Negative Takata-Ara	Positive Gum Mastic and Negative Takata-Ara	Negative Gum Mastic and Positive Takata-Ara	Percentage of Disparity
Dementia paralytica	47	39	0	8	0	17
Senile dementia and arteriosclerotic brain disease	10	1	1	1	7	75
Other organic brain disease	9	1	3	1	4	55
Alcoholic psychoses	47	9	11	4	23	41.7
Manic-depressive psychoses	16	3	8	1	4	31.7
Schizophrenia	31	4	12	1	14	48.4
Other psychoses	20	2	12	1	5	30
Totals	180	59	47	17	57	
Average disparity						43

fluid the mixture remains clear and violet. In a case of parasyphilis, the mixture becomes cloudy and a precipitate forms. Three grades of reaction are distinguished according to the promptness and quantity of the precipitation and the degree of clearness of the supernatant fluid. In the presence of meningitis, the fluid becomes red.

ANALYSIS OF RESULTS

In the accompanying table, the results in seven main clinical entities are recorded. By a negative gum mastic is meant a reading of either 1100000000 or 0000000000; a positive gum mastic includes everything from a low "syphilitic-zone curve" to one of the dementia paralytica type. To secure a greater reliability in these mastic readings as controls, all cases wherein such readings were nonconcomitant with the Pandy and Ross-Jones reactions were discarded from the tabulations. For this reason these common globulin tests are not included in this table.

4. Cockrill, J. R.: Comparison of Gold Chloride, Benzoin, and Mastic Tests on Cerebrospinal Fluid, Arch. Neurol. & Psychiat. **14**:455 (Oct.) 1925.

A glance at the results will make evident the pronounced disagreements in the results of the two tests involved, particularly in the case of arteriosclerotic and senile brain disease and other organic conditions. A more careful analysis shows the greatest disparity to be in the column headed, "Negative gum mastic and positive Takata-Ara." Even in the fluids of patients with alcoholic psychoses and schizophrenia there was a surprisingly large number of positive reactions with the Takata-Ara test—results which certainly are not in keeping with experiences with other colloidal reactions.

In thirty-nine of forty-seven cases of dementia paralytica, the gum mastic and the Takata-Ara reactions were positive; in the remaining nine fluids, the gum mastic showed the typical curve of dementia paralytica but the Takata-Ara reaction was practically negative. This means that in 17 per cent of this series the test failed to detect the protein fraction of dementia paralytica which was demonstrated by the more familiar globulin tests, and the results differ from those of Blum, who found a positive reaction in 98 per cent of his cases of dementia paralytica.

In sharp contrast to these observations, in ten cases of unquestionable arteriosclerotic brain disease seven spinal fluids were positive to the Takata-Ara reaction when the gum mastic readings were entirely negative. This would tend to offset the claim that the Takata-Ara reaction is highly specific for syphilis, and, on the other hand, would establish the belief that it also responds to a protein fraction associated with retrogressive arteriosclerotic changes. In other organic brain diseases, such as brain tumor, cerebral trauma and embolism, the results again suggested the possibility that the Takata-Ara test was sensitive to protein elements other than those of syphilitic brain disease. In this group is included one case of tuberculous meningitis in which the Takata-Ara test resulted in a mixed reaction.

In the various forms of alcoholic psychoses, there is a striking tendency for the Takata-Ara reaction to furnish positive reactions in the presence of negative gum mastic readings. Of a total of forty-seven such cases, twenty-three of the spinal fluids gave positive results with the Takata-Ara test when the other colloidal reactions were negative.

In the face of the fact that in the primary psychoses, namely, manic-depressive psychosis and schizophrenia, the familiar spinal fluid reactions are rarely positive, the Takata-Ara test very frequently showed positive results. It is true that many of these were positive only when the readings were delayed for from twelve to twenty-four hours. Fourteen of thirty-one spinal fluids from schizophrenic patients produced a cloudy precipitate with the Takata-Ara reagents after all the other protein tests were found to be negative.

COMMENT

It may be pointed out that mercuric chloride is the active precipitating reagent in the Takata-Ara test and the same substance is the reagent of the older Weichbrodt test. Experience with this latter reaction, most authorities will agree, has not borne out Weichbrodt's contention that it is a reliable specific test for syphilitic brain disease. The "bluish cast" mentioned by its author as evidence of syphilis has been noted in many nonsyphilitic conditions, and the Weichbrodt test is at the present time not part of the routine examination of the spinal fluid in most laboratories. Again, mercuric chloride is a reagent in Levinson's differential precipitation test for the meningitides. Here it is significant in detecting the proteins of tuberculous meningitis. From these considerations it must be apparent that mercuric chloride cannot claim a high degree of specificity for any particular pathologic process in the cerebrospinal tissues.

CONCLUSIONS

From clinical experience with the Takata-Ara test we draw the following conclusions:

1. The test is not highly specific. While it is positive in about 82 per cent of the cases of metasyphilis, there is enough disagreement to make its value as a routine test in syphilis rather doubtful.

2. As noted by other workers, it is very frequently postive in other organic, nonsyphilitic conditions, such as cerebral arteriosclerosis, brain trauma and tumor.

3. Although, in comparison with other colloidal reactions, the test is simple to carry out and to read, it has no advantage over the Weichbrodt reaction.

HEREDITARY FACTORS IN MANIC-DEPRESSIVE PSYCHOSIS

A COMPARISON OF INSTITUTIONAL AND EXTRAMURAL CASES *

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In three previous communications based on a study of 633 cases of manic-depressive psychosis in the private practice of Dr. Hugh T. Patrick, I [1] showed that these cases differed materially from this disorder as described in the literature. These differences were principally the presence of many attacks lasting from only a few hours to a few days, practically equal sex distribution and a tendency to a much later onset, shorter attacks and longer intervals. The existence of these differences was explained by the fact that the literature on manic-depressive psychosis has been developed almost entirely by physicians working in institutions for the insane; they saw only institutional cases, that is, the most malignant forms, and based their descriptions on these. In Dr. Patrick's material there were many cases so mild that hospitalization was not necessary, a type of case that physicians in institutions seldom saw and hence did not describe.

Since hereditary factors play an important rôle in the etiology of this disorder, the question arose as to whether the differences noted were correlated with differences in the hereditary elements. Do the milder extramural cases show a different heritage from that of hospital patients? The present study is concerned with this question.

Older writers recognized a hereditary tendency in cases of mania or circular insanity. Kirn [2] found evidence of a hereditary neuropathic taint in 76.1 per cent, Mendel [3] in 53 per cent, Karrer [4] in 60 per cent,

* Submitted for publication, Feb. 18, 1930.

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1. Paskind, H. A.: Brief Attacks of Manic-Depressive Depression, Arch. Neurol. & Psychiat. **22**:123 (July) 1929; Manic-Depressive Psychoses as Seen in Private Practice: Sex Distribution and Age Incidence of First Attacks, ibid. **23**: 152 (Jan.) 1930; Manic-Depressive Psychosis in Private Practice: Length of the Attack and Length of the Interval, ibid. **23**:789 (April) 1930.

2. Kirn, L.: Die periodischen Psychosen, Stuttgart, Ferdinand Enke, 1878.

3. Mendel, E.: Die Manie, Vienna and Leipzig, Urban & Schwarzenberg, 1881.

4. Karrer, F.: Bemerkungen zur zirkulären Geistesstörung, Allg. Ztschr. f. Psychiat. **37**:695, 1881.

Mordret [5] in 62.2 per cent, Lewis [6] in 36 per cent, Levin [7] in 58 per cent and Willerding [8] in 41.3 per cent. Hoche [9] stated that the disease developed almost exclusively on the basis of heredity. With the turn of the present century, there were published data pertaining to the hereditary factors which have not been materially changed to this day. Fitschen [10] found evidences of a hereditary taint in 80.8 per cent of cases, Weygandt [11] in 90 per cent, Ziehen [12] in 80 per cent, Soukanoff and Gannouchkine [13] in 87.5 per cent, Peixotto [14] in from 60 to 80 per cent, Saiz [15] in 84.7 per cent, Strohmayer [16] in from 75 to 80 per cent, Kraepelin [17] in 80 per cent, Durand [18] in 80 per cent, Bleuler [19] in 80 per cent, Pilcz [20] in 80 per cent, Sünner [21] in 84.13 per cent and Lange [22] in 90 per cent.

In my series the heredity was noted in 485 cases. In 83.1 per cent there was a definite history of a familial neuropathic tendency. In this respect the milder private cases do not differ essentially from the institutional cases described in the modern literature.

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5. Mordret, A. E.: De la folie à double forme, Paris, J. B. Baillière et fils, 1883.

6. Lewis, W. B.: A Textbook of Mental Diseases, Philadelphia, P. Blakiston's Son & Company, 1890.

7. Levin, H.: Ueber periodische Manie, Strassburg, C. Goeler, 1890.

8. Willerding, W.: Prognose bei Manie, Allg. Ztschr. f. Psychiat. **48**:72, 1892.

9. Hoche, A.: The Milder Forms of Periodical Insanity, Alienist & Neurol. **19**:193 (April) 1898.

10. Fitschen, E.: Die Beziehung der Heredität zum periodischen Irreseins, Monatschr. f. Psychiat. u. Neurol. **7**:127, 1900.

11. Weygandt, W.: Ueber das manisch-depressive Irresein, Berl. klin. Wchnschr. **38**:70, 1901.

12. Ziehen, T.: Psychiatrie, ed. 2, Leipzig, S. Hirzel, 1902.

13. Soukanoff, S., and Gannouchkine, P.: Étude sur la manie, Arch. de neurol. **15**:40 (May) 1903.

14. Peixotto, A.: Folie maniaque dépressive, Ann. méd. psychiat. **1**:202, 1905.

15. Saiz, G.: Untersuchungen über die Aetiologie der Manie, der periodischen Manie, und des zirkulären Irreseins, Berlin, S. Karger, 1907.

16. Strohmayer: Manisch-depressive Irresein, Wiesbaden, F. C. W. Vogel, 1914.

17. Kraepelin, E.: Manic-Depressive Insanity and Paranoia, translated by Mary Barclay, Edinburgh, E. & S. Livingstone, 1921.

18. Durand, M., in Sergent, Ribadeau-Dumas and Barbonneix: Traité de pathologie médicale et de therapeutique appliqué, Paris, A. Maloine et fils, 1921.

19. Bleuler, E.: Textbook of Psychiatry, translated by A. A. Brill, New York, The Macmillan Company, 1924.

20. Pilcz, A.: Lehrbuch der speziellen Psychiatrie, Leipzig and Vienna, Franz Deuticke, 1926.

21. Sünner, P.: Die psychoneurotische-erbliche Belastung bei dem manisch-depressiven Irresein, auf Grund der Diem-Kollerschen Belastungsberechnung, Ztschr. f. d. ges. Neurol. u. Psychiat. **77**:453, 1922.

22. Lange, J., in Bumke, Oswald: Handbuch der Geisteskrankheiten, Berlin, Julius Springer, 1928, vol. 6.

The best data that I could find regarding the neuropathic factors in manic-depressive psychosis are those of Sünner.[21] He compared these factors in 650 cases of manic-depressive psychosis from the Munich clinic with similar factors in the sane (after Diem), in psychotic patients in general (after Koller and Diem), in patients with dementia paralytica (after Kalb) and in those with epilepsy (after Snell). In the accompanying tables I have arranged my data so that comparisons can be made with Sünner's institutional cases. For the sake of ready comparison, I have also tabulated corresponding data for the sane (Diem [23]) and for the insane in general (Diem [23] and Koller [24]).

In the present series a neuropathic factor could be detected in the direct line (parents) in 67.9 per cent of the cases, in the indirect line only (grandparents, uncles and aunts) in 6.2 per cent, and in siblings only in 9.1 per cent. In table 1, I have given a comparison of these data

TABLE 1.—*Percentage of Cases in the Sane, the Psychotic in General, Institutional Patients with Manic-Depressive Psychosis and the Present Series, in Which a Neuropathy Occurs in the Direct Line (Parents), Indirect Line (Grandparents, Uncles and Aunts) Only, and Siblings Only*

	Sane (Koller)	Psychotic in General		Manic-Depressive Psychosis	
		Koller	Diem	Institutional (Sünner)	Extramural (This Series)
Direct	33	50	57	65.53	67.9
Indirect only	29	12	16	14.76	6.2
Siblings only	5	7	9	4.76	9.1
Total	67	69	82	85.05	83.2
		Average 76			

with similar data for institutional cases, for the sane, and for the psychotic in general. This table shows that as regards neuropathy in the direct line, Sünner's institutional series and the present one are in close accord—65.5 per cent and 67.9 per cent, respectively. In the number of cases in which neuropathy was found in the indirect line only and in siblings only, there is a difference. It may also be noted that taint in the direct line is much less frequent in sane (33 per cent) and in psychotic subjects in general (from 50 to 57 per cent).

In table 2 I have shown the nature of the neuropathic factor in the direct line in the present series and have given a comparison between it and similar data for the sane, for psychotic persons in general and for institutional patients with manic-depressive psychosis. In my series a

23. Diem, O.: Die psycho-neurotische-erbliche Belastung der Geistesgesunden und der Geisteskranken, Arch. f. Rassen- u. Gesellsch. **2**:215, 1905.

24. Koller, J.: Beitrag zur Erblichkeitsstatistik der Geisteskranken in Canton Zurich, Vergleichung derselben mit der erblichen Belastung gesunder Menschen durch Geistestörungen, Arch. f. Psychiat. u. Nervenk. **27**:268, 1895.

psychosis was found in the parents in 23.3 per cent of cases, a nervous disorder in 40.7 per cent, alcoholism in 2.8 per cent, psychopathic personality in 0.7 per cent and suicide in 1.2 per cent. It may be noted that a psychosis in the direct line occurs in the present series more frequently than in Sünner's cases (18.92 per cent). In my series psychosis in the direct line was ten times as frequent as in the sane and 5.1 per cent more frequent than in insane persons in general. I found nervous disorders much more frequent (40.7 per cent) than did Sünner, but in the present series alcoholism, psychopathic personality and suicide were less frequent than in Sünner's institutional cases. Apoplexy and senile dementia were considered by Sünner, but in the present series such data were not available. It is possible that the discrepancy between the figures for psychoses in the direct line in Sünner's series and in this one is due to the fact that senile dementia is included in my figures and not in

TABLE 2.—*Percentage of Cases in the Sane, the Psychotic in General and Institutional and Extramural Patients with Manic-Depressive Psychosis in Which the Various Neuropathic Taints Occurred in the Direct Line (Parents)*

	Psychosis	Nervous Disorders	Alcoholism	Apoplexy	Senile Dementia	Psychopathic Personality	Suicide
Sane (Diem)	2.2	5.7	11.2	5.9	1.4	5.9	0.4
Psychotic in general (Koller and Diem)	18.2	5.0	13-21	3.2	1.6	8-13	0.5-1.0
Institutional patients with manic-depressive psychosis (Sünner)	18.92	14.0	11.38	8.15	2.15	9.38	1.53
This series	23.3	40.7	2.8	....	....	0.7	1.2

Sünner's. The differences in the frequency of alcoholism and psychopathic personality may be explained partly by the fact that in private practice the patients are from a higher social stratum. The figures for suicide in the present series correspond well with those of Sünner, 1.2 per cent and 1.53 per cent, respectively, and are greater than those for the parents of sane persons (0.4 per cent) and for the parents of psychotic subjects in general (from 0.5 to 1 per cent).

THE NATURE AND DISTRIBUTION OF THE NEUROPATHIC FACTORS

In the present series, the 485 patients had 877 relatives exhibiting evidences of a neuropathy. The latter had the following distribution:

Manic-depressive psychosis, 216; psychosis, type not determined, 91; nervousness, 325; psychoneurosis, 5; migraine, 166; epilepsy, 10; erratic or eccentric behavior, 8; writer's cramp, 2; spasmodic torticollis and tic, 4; feeblemindedness, 2; alcoholism, 26; psychopathic personality, 3; suicide (no psychosis mentioned), 19.

Expressing these figures in terms of percentage, it was found that of the 877 neuropathically tainted persons 35.0 per cent had a psychosis

and 59.5 per cent a nervous disorder (i. e., nervousness, psychoneurosis, migraine, epilepsy, eccentricity, writer's cramp, spasmodic torticollis, tic, feeblemindedness [25]), 2.9 per cent suffered from alcoholism, 0.3 per cent had a psychopathic personality and 2.1 per cent were suicides. How the foregoing data compare with similar data for the sane, the psychotic in general and hospitalized patients with manic-depressive psychosis is shown in table 3. A study of this table elicits some interesting data. While in the families of sane subjects 15.1 per cent, and in the psychotic in general 45.9 per cent, of tainted relatives had a psychosis, in institutional cases of manic-depressive psychosis this factor was 40.2 per cent, higher than in the sane and lower than in the insane in general. In the present series this factor was 35 per cent. It was less than Sünner's value for the same factor, but agreed with that of Sünner in that it exceeded the percentage found in sane subjects and was less than that

TABLE 3.—*Distribution of Neuropathy Among the Relatives of the Sane, the Psychotic in General, Institutional Patients with Manic-Depressive Psychosis and This Series*

	Psychosis	Nervous Disorders	Alcoholism	Apoplexy	Senile Dementia	Psychopathic Personality	Suicide
Nonpsychotic (Diem) 1,945 factors	15.1	14.9	21.3	18.4	7.4	21.3	1.6
Psychotic (Koller and Diem) 3,515 factors	45.9	5.1	20.9	5.5	1.4	18.8	2.3
Institutional patients with manic-depressive psychosis (Sünner) 1,199 factors	40.2	22.2	9.0	10.8	2.8	10.6	4.4
This series, 877 factors	35.0	50.5	2.9	....	....	0.3	2.1

found in psychoses in general. I found nervous disorders to be much more frequent than did Sünner.[26] Alcoholism and psychopathic personality were decidedly less common than in Sünner's cases; instances of suicide were slightly less than half as numerous as Sünner found.

CONCLUSIONS

Since it was shown previously that extramural cases of manic-depressive psychosis present important differences from the institutional cases described in the literature, an attempt was made to determine whether these differences were correlated with a different heredity. Four hundred and eighty-five extramural cases of manic-depressive psychosis

25. Defective mental development is, strictly speaking, neither a psychosis nor a nervous disorder, but for the sake of convenience it is included with the nervous disorders.

26. Sünner and Diem considered neurasthenia, nervousness, hysteria, epilepsy, traumatic neuroses, fear neuroses, migraine, chorea, enuresis and paralysis agitans among the nervous disorders.

were studied from the standpoint of hereditary tainting factors, and the data were compared with those in institutional cases. In the extramural cases a hereditary taint could be demonstrated in 83.2 per cent, a figure corresponding well with that of all modern writers for institutional cases. In the direct line a neuropathic taint was found in 67.9 per cent of extramural cases; this value is very close to the similar value for institutional cases. In the direct line, psychoses and nervous disorders were more frequent in extramural patients; in the latter, alcoholism, psychopathic personality and suicide were less frequent. All tainted relatives were considered and compared in the two series: Psychosis was found somewhat less frequently in the extramural cases; nervous disorders were more common in the private cases, while alcoholism, psychopathic personality and suicide were less frequent in the extramural cases.

This study lends support to the opinion that the very mild and very brief cases of manic-depressive depression, frequently not recognized as such, are in nature (fundamentally, nosologically) identical with the classic institutional cases.

INFLUENCE OF INTERCURRENT FEBRILE DISORDERS ON PREEXISTING EPILEPSY *

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Improvement in cases of epilepsy during and following acute infectious diseases has been reported in many individual cases. The relatively small number reported by any one observer has led me to review 200 cases of epilepsy in patients of all ages in which records of convulsions had been kept for several months previous to, and for a similar time after, some acute febrile disorder.

The diseases of childhood have been credited with many cures of epilepsy, but such cures may have been coincidental recoveries. There are two periods of life wherein the greatest number of cases of epilepsy develop; [1] namely, the first four years of life, and at puberty. Singularly, these are also the periods of life during which most cures of epilepsy are reported. There are logical explanations of these recoveries which are entirely independent of any intercurrent disease. During these periods the most marked physiologic changes occur, and many idiosyncrasies become manifest.

Wallis, Mackenzie and Nicol [2] found that certain young epileptic patients who did not respond to bromides were hypersensitive to certain proteins immediately before convulsions but became desensitized for a time following seizures. They tested 122 epileptic patients and found 46 sensitive to proteins. These authors believed that the treatment in certain selected cases of epilepsy was essentially the treatment for food idiosyncrasy. They believed migraine and some cases of epilepsy were associated with protein hypersensitivity. Edgeworth [3] treated 20 patients with epilepsy by intravenous injections of 5 per cent peptone. He reported temporary arrest of convulsions in nine cases and diminution in frequency in 6 cases.

The recent success of fever therapy in dementia paralytica has stimulated research into the effect of similar treatment for epilepsy.

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* Submitted for publication, Feb. 17, 1930.

* From the Monson State Hospital.

1. Partridge, J. C.: Epilepsy and the Endocrines, Am. J. Psychiat. **8**:137 (July) 1928.

2. Wallis, R. J.; Mackenzie, and Nicol, W. D.: The Importance of Protein Hypersensitivity in the Diagnosis and Treatment of Epilepsy, Lancet **1**:741, 1923.

3. Edgeworth, F. H.: Intravenous Protein Therapy in Epilepsy, Brit. M. J. **2**:780, 1920.

Arias [4] quoted Aguglia and D'Abundo, who reported four cases of epilepsy in which the patients were treated by malarial inoculation. In these patients, who had been having daily convulsions, the seizures disappeared completely during the course of the fever, and improvement was noticeable for three months following this treatment. The same author selected two cases which had been very refractory to phenobarbital, sodium borotartrate, and potassium bromide therapy, and inoculated the patients with malaria. They had a daily rise of temperature for twelve days, during which they had no convulsions, and they did not have any seizures for two months although they received no medication. After two months they began to have convulsions again, but they were less refractory to medical treatment. The author then treated twelve patients with epilepsy by malarial inoculation, with improvement in eight cases. He believed that malaria therapy had virtue when used in selected cases of epilepsy, and thought that patients who did not respond to ordinary medical treatment offered the best results. His conclusions were that patients so treated relapsed, but convulsions were less frequent and offered less resistance to ordinary medical treatment.

Mendel [5] reported two cases of epilepsy following attacks of malaria in adults, and concluded that malaria might cause organic changes in the central nervous system which result in the epileptic state. He believed that epilepsy was sometimes a definite sequel of malaria. His report tends to discourage one in attempting this type of fever therapy in epilepsy.

Hamilton [6] stated that the effect of typhoid fever on epilepsy was uniformly favorable during the febrile period. He also reported a case of vaccination with staphylococcus culture, with marked mental improvement lasting two weeks.

Clark and Sharp [7] observed several epileptic patients with intercurrent diseases and found only two cases in which epilepsy was notably improved, but he saw many epileptic patients who were made worse by infections. The improvement in two cases was only temporary and occurred during and immediately after measles.

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4. Arias, B. Rodriquez: Effects of Malaria Therapy in Essential Epilepsy, Rassegna internaz. di clin. e terap. **9**:673, 1928.

5. Mendel, K.: Epilepsy Following Malaria, Med. Klin. **22**:1770 (Nov. 12) 1926.

6. Hamilton, A. S.: Effect of Intercurrent Disorders on Preexisting Epilepsy, J. A. M. A. **53**:1902 (Dec. 4) 1909.

7. Clark, L. P., and Sharp, E. A.: Influence of Measles and Erysipelas upon Epilepsy, Med. News **77**:853, 1900.

Maillard [8] reported a 75 per cent decrease in convulsions during attacks of influenza, but the frequency of seizures returned to normal after the fever subsided. He did not give the number of cases on which his observations were based.

Gordon [9] studied twenty-five cases of epilepsy complicated by influenza. He reported complete suppression of convulsions during the febrile period. As soon as the febrile state began to subside, there was a tendency to the return of convulsions; however, the attacks were lessened in severity for some time after the temperature became normal. The author believed that during an infectious state a toxic principle was elaborated which was capable of neutralizing toxic principles existing in the blood of epileptic patients. He concluded that serum therapy was indicated, and that the problem was to produce such a neutralizing biologic product. Gordon apparently did not consider the psychic effect of an acute illness on the course of seizures or the possible effect of dehydration or acidosis, either of which might develop during the course of an acute febrile disorder.

Muskens [10] believed that the initial manifestation of epilepsy frequently follows scarlet fever, pneumonia, influenza, malaria and septic disorders; however, he stated that the person with chronic epilepsy has a temporary alleviation from the malady if he has an acute illness.

Menninger [11] reported a limited number of cases of epileptic patients who showed mental improvement during attacks of influenza. The improvement in some of his cases apparently was prolonged after the acute febrile period.

Favorable results in cases of epilepsy following venesection, various operative procedures and emotional shock have been reported. In the Monson State Hospital research workers with cameras or other instruments, intent on obtaining data during convulsions, observed patients for long periods of time without being favored with a single explosion; however, under ordinary circumstances these patients had several convulsions each day.

Reviewing the literature, one finds general agreement that convulsive seizures in epileptic subjects are decreased in number during the course of febrile disorders, but there is no agreement that preexisting epilepsy is favorably modified for any length of time after the fever

8. Maillard, M. G.: Influenza and Epilepsy, Am. J. Clin. Med. **30**:798 (Nov.) 1923.

9. Gordon, A.: Influenza and Epilepsy, Med. Rec. **40**:942, 1921.

10. Muskens, L. J. J.: Epilepsy, ed. 1, New York, William Wood & Company, 1928, p. 284.

11. Menninger, Karl A.: The Amelioration of Mental Disease by Influenza, J. A. M. A. **94**:630 (March 1) 1930.

subsides. Epileptic patients are predisposed to circulatory failure, and their general vulnerability calls for caution.

In the 200 cases of epilepsy here reviewed, the patients had the following febrile disorders while in this hospital: influenza, 85; scarlet fever, 17; mumps, 17; pneumonia, 16; measles, 14; chickenpox, 14; erysipelas, 11; tonsillitis, 11; diphtheria, 9, and whooping cough, 6. The average period of observation before the month of acute illness, and after the month of acute illness, was 10.6 months. To facilitate a review of the record of convulsions, the entire month during which the acute illness occurred was tabulated separately, and the number of convulsions during this month of acute illness was compared with the average monthly rate of convulsions during 10.6 months prior to and 10.6 months following the month of acute illness. The accompanying table

Analysis of Two Hundred Cases Showing Relative Frequency of Convulsions Before, During and After Acute Febrile Period

		Frequency of Convulsions After Acute Illness			Average Frequency per Month Prior to, During and After Acute Illness		
	Cases	Increase	Unchanged	Decrease	Prior to	During	After
Influenza	85	38	3	44	6.17	6	6.8
Scarlet fever	17	12	0	5	10.81	4.76	11.37
Mumps	17	6	2	9	5.25	5	5.36
Pneumonia	16	8	0	8	5.34	5.43	5.74
Measles	14	7	1	6	3.12	3.53	3.92
Chickenpox	14	6	1	7	13.3	5.4	4.68
Erysipelas	11	4	0	7	4.77	4.6	4.3
Tonsillitis	11	5	0	6	4.51	3.9	4.45
Diphtheria	9	2	0	7	3.85	0.55	3.34
Whooping cough	6	2	0	4	5	6.5	5.2
Total	200	90	7	103	6.39	5.12	6.16

represents an analysis of the number of convulsions with relation to the acute diseases mentioned.

The series of cases of each disease is too small to be significant; however, the group taken as a whole gives a representative cross-section of the influence of febrile disorders in epilepsy. Among 85 patients with influenza, slightly over one-half showed a decrease in the number of convulsions following this disease; however, the total number of convulsions in all cases was slightly increased. Among 17 patients with scarlet fever, a great majority had more convulsions after the acute illness than before, and the total number of convulsions increased following the acute infection; however, the decrease in number of seizures during the month of acute illness was most noticeable. After chickenpox, erysipelas, tonsillitis and diphtheria, there was a slight decrease in the number of convulsions, but the number of patients showing this relative improvement was so limited that these apparently favorable results are considered practically negligible. The decrease in the num-

ber of seizures which occurred in 200 cases followed over 10.6 months before and 10.6 months after an acute febrile disorder was only 3.6 per cent. After considering the various conditions which ordinarily influence the frequency of convulsions over such a period of time, one is inclined to discount this small change. In the Monson State Hospital a gradual decrease has been noted in the frequency of seizures in patients showing progressive deterioration with definite neurologic signs. This probably offsets the almost negligible decrease in seizures (3.6 per cent) following acute febrile states.

The most prominent feature of this analysis is the decrease in the number of seizures in most cases during the month of illness, and this is reflected in the comparison of total averages before, during and after the acute illness. The decrease in the number of seizures during the month of illness, as compared with the average monthly rate of convulsions for 10.6 months before and 10.6 months after the acute illness, amounts to 18.5 per cent.

By grouping the respiratory diseases together (107 cases of influenza, pneumonia and whooping cough), the change in frequency of seizures is found to be very slight. In these 107 cases the average monthly rate of convulsions previous to the acute illness was 5.9, during the month of acute illness 5.9, and following the acute illness 6.4. This gives an increase of 7.8 per cent after the febrile period and no decrease in convulsions during the month of illness as compared with previous months. The changes, then, are found chiefly among cases of non-respiratory diseases, scarlet fever, mumps, measles, chickenpox, erysipelas, tonsillitis and diphtheria. In this group, composed of 93 cases, the monthly rate of convulsions previous to the acute illness was 6.9, during the month of acute illness 4.2, and following the month of acute illness, 5.7. These figures give a decrease of 17 per cent following the acute illness as compared with the rate before the acute illness. In this group there is also a decrease in seizures of 33.33 per cent during the month of acute illness as compared with the average rate before and after the acute disease. This change calls for explanation, because the decrease may be more real than apparent, as one expects patients to be more carefully watched during an acute illness, and all seizures are recorded more faithfully. Although the number of cases in these groups is too small for one to arrive at any conclusions, the results clearly indicate that the favorable influence of fever occurred in patients without acute respiratory infections.

It is hard to reconcile these observations with the fact that certain patients, especially children, have seizures only at the time of acute infection. Naturally, such children were not included in the group studied, because, for purposes of comparison, cases were selected in

which a fairly regular epileptic habit had been established. Lennox[12] does not believe that the degree of acidosis caused by these febrile disorders would influence the results unless the patients were either partially fasting or dehydrated during this time. A limited diet and limited fluid intake, resulting in moderate acidosis, may explain the decrease in convulsions in the course of febrile disorders. Patients who are acutely ill receive more careful attention, especially with regard to diet and elimination, and this, together with some changes in the emotional state, may explain these observations; however, one looks for a direct biochemical solution.

12. Lennox, William G.: Personal communication.

Clinical Notes

A CASE OF ENCEPHALITIS AND INFLUENZAL PNEUMONIA FOLLOWING VACCINATION*

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The case of encephalitis which is here described was diagnosed clinically as pneumonia and encephalitis. When at autopsy it was found that the pneumonia was an influenza bacillus bronchopneumonia, it seemed probable that the encephalitis was the so-called influenzal encephalitis. Later it was learned that the child had been vaccinated one week before the onset of illness.

The encephalomyelitis which sometimes follows vaccination for variola has been only recently added to the number of inflammations of the brain and cord that have been studied. The inflammatory reaction is similar to that found in encephalitis following measles, typhoid, pertussis and varicella and in rabies. The infiltrating round cells are found not only around the veins, but spread out on the brain substance. There is also an infiltration of the pia-arachnoid of the cerebrum. In the cerebrum and cerebellum the white substance is chiefly affected. In the brain stem and cord the gray substance is more affected than the white, but it is not the gray substance around the ventricles and sylvian aqueduct that is infiltrated.

The situation in regard to the so-called influenza encephalitis is one of confusion, and is further complicated by a variable nomenclature. Since 1850, encephalitis has been assumed to be related to influenza. In 1890, Leichenstern [1] made the first histologic examination of the brain in cases considered to be influenza encephalitis or encephalitis hemorrhagica. He described a purulent meningitis and numerous areas of hemorrhagic softening in the cortex. Fürbringer,[2] in 1892, reported two cases of encephalitis hemorrhagica. In the brain in one subject there were multiple punctate hemorrhages, but no microscopic examinations of the brain were reported. In the second case there was an egg sized hemorrhagic area of softening in the central gyrus. Nauwerck,[3] in 1895, published a report of a case in which Pfeiffer's bacillus was recovered from a large apoplectic area in the cerebellum. In another case of his there were multiple areas of softening from a sinus thrombosis, but no bacilli were found. Pfuhl [4] also reported the isolation of Pfeiffer's bacillus in cases of influenzal encephalitis, but the bacilli were found in a purulent meningitic exudate. In 1896, Leichenstern [5] classified all the cases cited, together with primary acute hemorrhagic encephalitis and nona as influenzal encephalitis.

* Submitted for publication, April 1, 1930.

* From the Pathological Laboratory of the Buffalo General Hospital.

1. Leichenstern, O.: Deutsche med. Wchnschr. **16**:510, 1890.

2. Fürbringer, O.: Deutsche med. Wchnschr. **18**:45, 1892.

3. Nauwerck, F.: Deutsche med. Wchnschr. **21**:393, 1895.

4. Pfuhl, A.: Deutsche med. Wchnschr. **45**:457, 1919.

5. Leichenstern, O., in Nothnagel: Specielle Pathologie und Therapie, Vienna, 1896.

Primary acute hemorrhagic encephalitis and nona are identical and are now known as epidemic encephalitis. Oppenheim [6] considered the cases cited of influenzal encephalitis as simple cerebral softening—encephalomalacia—rather than encephalitis. He gave no specific information, but he thought that encephalitis might be caused by influenza. Schmidt,[7] Chartier [8] and Comby [9] held similar opinions. An approach to the relation of encephalitis and grip is found in a case reported by Ulrich [10] in 1911. A mother who had nursed her children through influenza became ill with headache, loss of appetite and drowsiness. She died in six days. There was found a round cell infiltration of the vessels of the gray substance of the brain stem and thalamus. The microscopic observations appeared to be similar to those which Economo [11] found in the cases described by him under the name of encephalitis lethargica in 1917. In the many publications which followed Economo's study of this disease, a number of authors considered that encephalitis lethargica and encephalitis of influenza were identical. This opinion was influenced not only by the previous association of encephalitis with influenza, but by the then prevalent (1917-1918) epidemic of influenza.

Economo,[12] in 1919, attempted to distinguish between encephalitis lethargica and encephalitis grippalis by publishing two cases of what he called encephalitis hemorrhagica or the encephalitis of influenza. In one case, multiple abscesses of the lungs and other organs were found. In the brain there was one large hemorrhage in the occipital lobe and beginning generalized miliary abscesses. There was no infiltration of the blood vessels. Högler [13] also reported a case of so-called encephalitis grippalis, in which there were areas of hemorrhagic softening. Neither of these cases should be classified as encephalitis, but as encephalomalacia, the first as pyo-encephalomalacia.

The patient in Economo's second case developed signs of encephalitis two weeks after "grip," and he died three weeks later. Histologically, there were capillary hemorrhages in the brain but no inflammatory reaction. This was designated "the toxic encephalitis of grip." Since there was no inflammation, it can scarcely be classified as encephalitis. The reports of ten similar cases were published by Marcus,[14] in 1918, as encephalitis of influenza. Juhl [15] and Wiesner [16] described such capillary hemorrhages as influenzal encephalitis or encephalitis hemorrhagica. Many others also reported hemorrhagic encephalitis solely from gross examination of the brain. Hart [17] and Siegmund [18] found similar capillary hemorrhages, but believed that the condition was not only "toxic encephalitis," but purpura cerebri. Schmidt,[7] in 1905, first described purpura cerebri in various diseases and cerebral trauma. He found areas of necrosis surrounding capillaries and small vessels.

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6. Oppenheim, H., in Nothnagel: Specielle Pathologie und Therapie, Vienna, 1896.
7. Schmidt, M. B.: Beitr. z. path. Anat. u. z. allg. Path. **7**:419, 1905.
8. Chartier, M.: Encéphale **2**:67, 1907.
9. Comby, J.: Arch. de méd. d. enf., 1907, no. 10, p. 45.
10. Ulrich, M.: Med. Klin. **7**:817, 1911.
11. Economo, C.: Wien. klin. Wchnschr. **30**:581, 1917.
12. Economo, C.: Wien. klin. Wchnschr. **32**:393, 1919.
13. Högler, F.: Wien. klin. Wchnschr. **33**:144, 1920.
14. Marcus, H.: Berl. klin. Wchnschr. **55**:1151, 1918.
15. Juhl, D.: Virchows Arch. f. path. Anat. **232**:58, 1921.
16. Wiesner, R.: Wien. klin. Wchnschr. **31**:1101, 1918.
17. Hart, C.: Encephalitis with and Following Influenza, Med. Klin. **16**:846 (Aug.) 1920.
18. Siegmund, F.: Berl. klin. Wchnschr. **56**:509, 1920.

These areas were walled off by large cells and red blood cells and an occasional leukocyte. In some cases he found only simple capillary hemorrhages, as compact bullet bleeding. He believed that purpura cerebri was produced by the effect of toxins on the blood vessels and was not an encephalitis. Later writers have held the same opinion. Mittasch,[19] however, considered purpura cerebri an inflammatory change. He linked it with epidemic encephalitis and believed them both to be of influenzal origin, two types of the same disease. There are essential differences in the histopathology of cerebral purpura and epidemic encephalitis. Purpura cerebri is found chiefly in the white substance and in no particular part of the gray substance, but it has no predilection for the gray substance around the ventricles and sylvian aqueduct. The clear zones of necrosis around the blood vessels are not found in epidemic encephalitis. Also, these areas of necrosis need not contain any cells and may be localized, which is similar to multiple sclerosis rather than to epidemic encephalitis. Lucksch [20] favored Mittasch's opinion that purpura cerebri and epidemic encephalitis are related. He thought that purpura cerebri is a toxic condition and not an inflammatory one, and occurs therefore in the early part of an influenza epidemic. As the virulence of the epidemic lessens, epidemic encephalitis makes its appearance. It is now known that epidemics of influenza are preceded and followed by epidemic encephalitis, which makes Lucksch's theory scarcely tenable. He suggested a further relationship between influenza and epidemic encephalitis in the similarity of the infiltrations of the bronchial walls and skin exanthems in influenza to the perivascular infiltrations in the brain in epidemic encephalitis. The infiltrations in epidemic encephalitis are similar to those of any other encephalitis.

The relation of influenza to epidemic encephalitis is a matter of scepticism to Achard,[21] Stern [22] and Wimmer.[23] As to influenzal encephalitis or encephalitis hemorrhagica, it is evident that there is no generally accepted concept of this disease. It is unfortunate that the name hemorrhagic encephalitis persists, since an encephalitis is inflammatory and not hemorrhagic. Also, the term is used indiscriminately for large apoplectic areas and capillary hemorrhages.

REPORT OF CASE

A. K., a boy, aged 6, was admitted to the Buffalo Children's Hospital on Dec. 21, 1928. The previous history and the family history were unimportant. On the first day of the boy's illness he complained of frontal headache which persisted for two days. He then became stuporous and incontinent. On the fourth day, after several convulsions, he was brought to the hospital. The chief physical observations were: lateral nystagmus and spasmodic twitching of the eyelids, cyanosis with stertorous breathing and tracheal râles, a patch of bronchial breathing in the left scapular region and tonic contraction of the extremities. On the upper part of the left arm was a small scab, probably the result of vaccination. There were 31,000 leukocytes per cubic millimeter of blood (87 per cent polymorphonuclear cells). The temperature was 102 F. The spinal fluid was under pressure (30 mm. of mercury).

19. Mittasch, G.: Frankfurt. Ztschr. f. Path. **26**:406, 1922.

20. Lucksch, F.: Pathologic Anatomy of Influenza, Arch. Path. **5**:448 (March) 1928.

21. Achard, P.: L'encéphalite léthargique, Paris, J. B. Baillière et fils, 1921.

22. Stern, F.: Ergebn. d. ges. Med., 1926, vol. 8.

23. Wimmer, A.: Chronic Epidemic Encephalitis, Copenhagen, Heinemann, 1924.

The fluid was clear and contained 12 cells per cubic millimeter. Pulmonary edema developed, and the patient died about eleven hours after admission to the hospital.

At autopsy, acute hemorrhagic bronchitis was found. There were multiple foci of bronchopneumonia of hemorrhagic character throughout the lungs. Smears from the bronchi showed gram-negative bacilli and a few gram-positive cocci. Smears from the lungs showed gram-negative rods resembling the influenza bacilli of Pfeiffer. There was acute hyperemia of the brain.

The microscopic sections of the organs showed nothing remarkable except influenzal bronchopneumonia in which Pfeiffer's bacilli could be demonstrated.

In the histologic examination of the brain, blocks were taken from the usual portions of the brain, fixed in alcohol and embedded in paraffin and celloidin. The

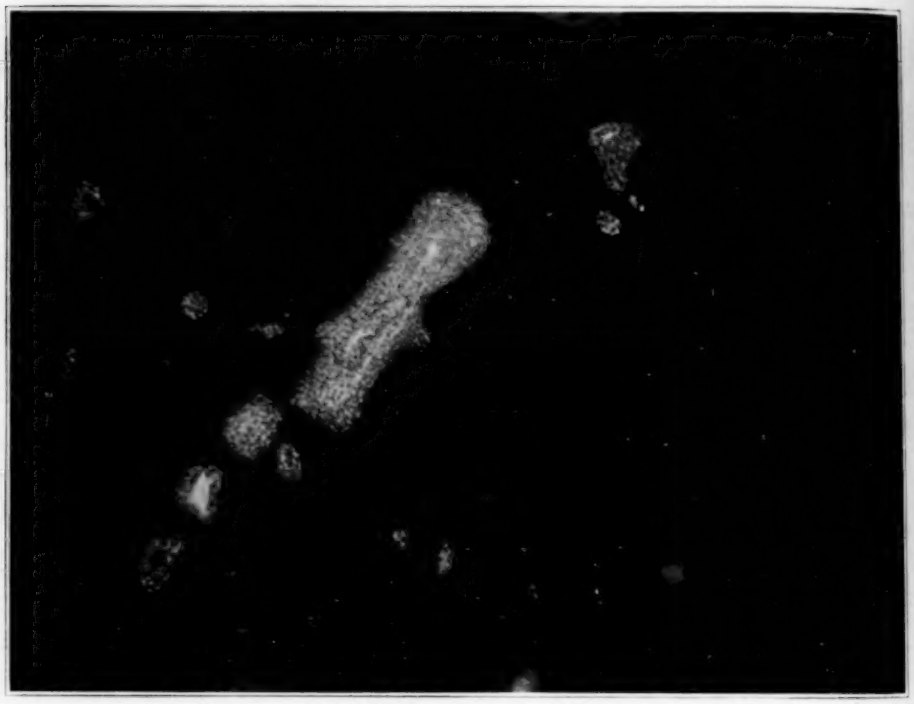

Fig. 1.—Infiltration of vessels and brain substance; thionine blue stain.

rest of the brain was fixed in formaldehyde. The stains used were thionine blue, hematoxylin-eosin, van Gieson, Herxheimer's fat stain, Cajal's glia stain, Holzer's glia fiber stain, Turnbull's blue stain for iron, methylene blue (methylthionine chloride, U. S. P.), Spielmeyer's myelin sheath stain and Weigert's elastic fiber stain. The meninges were neither infiltrated nor thickened. The architectonics of the cortex were not disturbed. The ganglion cells showed no characteristic changes. The glia cells, except in the vicinity of some of the vessels, were not enlarged or increased in numbers. Throughout the centrum ovale and the lower layer of the cortex there was a heavy cuffing of the blood vessels with small round cells, plasma cells and macrophages. The tissue surrounding these blood vessels in the centrum ovale was also infiltrated. The cells were similar to those in the adventitial spaces with the addition of microglia. In order to determine whether glia cells were a

part of the infiltration, Cajal and Holzer stains were made. In the Cajal preparations the cells in the infiltrated areas took a purplish color and no cells with processes could be seen, whereas fibrous glia cells were found up to the edge of these areas. The Holzer stain also showed no fibrous glia among the infiltrating cells. If any of these cells were glia, they were glia cells without processes. There did not appear to be any abnormal growth of glia surrounding the infiltrated areas. The precapillaries were the vessels chiefly infiltrated. It was difficult to determine whether the vessels were veins or arteries, since the infiltrating cells covered all the layers so that the Weigert and van Gieson stains did not show the elastic fibers and layer differentiation well. However, as some of the large arteries were infiltrated, it seemed probable that the smaller ones were also infiltrated.

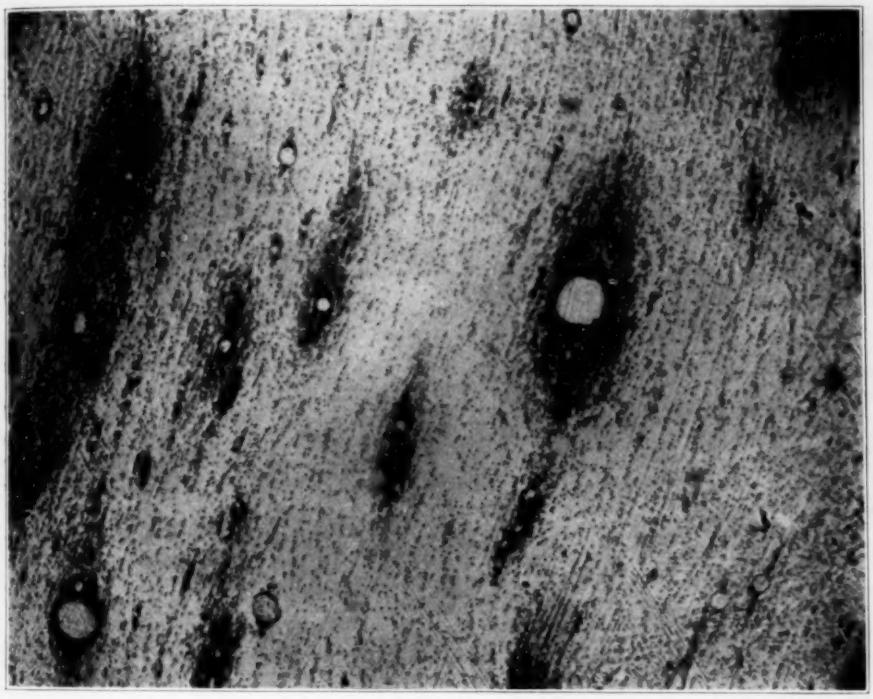

Fig. 2.—Clear spaces around infiltrated vessels; Spielmeyer's myelin sheath stain.

In the putamen and globus pallidus, as well as in the thalamus and nucleus caudatus, only a few vessels were cuffed with cells. The infiltrated vessels in the thalamus and caudate nucleus were confined to the lateral areas which bordered on the infiltrated white substance. Except for these areas in the thalamus and caudate nucleus, the nuclei around the ventricles and sylvian aqueduct were in no way disturbed. The only change in the substantia nigra was the presence of iron granules in the glia cells. In the pons, the white substance and its vessels were infiltrated, but comparatively less than in the centrum ovale. A few vessels of the pontile nuclei were slightly infiltrated. The white substance near the dentate nucleus contained several heavily cuffed vessels, but the rest of the cerebellum was free from infiltrations. There were no infiltrated vessels in the medulla or in

the cervical portion of the cord, the only part of the cord which was removed. By the Spielmeyer myelin sheath stain clear spaces were found around most of the infiltrated vessels. When compared with the hematoxylin-eosin stain of the same region, not all of the vessels showed a clear zone with the Spielmeyer stain, in spite of the fact that the surrounding tissue was also infiltrated. There were no fat globules in the infiltrated tissues or in any part of the brain tissue. There were no fat particles in the cells or lying loosely in the vessels which had an inflammatory infiltration. Fat globules were found in the adventitia of a few uninvolved vessels that lay beneath the gray substance of the cortex.

Thin paraffin sections of the brain showed no Pfeiffer's bacilli.

COMMENT

From the clinical history it is obviously impossible to distinguish between three forms of encephalitis, the so-called influenzal encephalitis, postvaccinal encephalitis and epidemic encephalitis. Influenzal encephalitis as an entity can be disregarded. The microscopic examination of the brain does not show an inflammatory reaction in the areas which are characteristic of epidemic encephalitis—the gray substance around the ventricles and the sylvian aqueduct. It is true that the thalamus and caudate nucleus are slightly infiltrated, but only in the lateral areas. The histopathologic observations correspond rather to those found in postvaccinal encephalitis. In the cerebrum, the white substance is chiefly affected as has been described in postvaccinal encephalitis. In the cases of Turnbull and McIntosh,[24] the basal ganglia were but slightly affected and the thalamus only in the lateral areas, as in my case. The greatest areas of infiltration have been described in the pons and upper medulla by Perdrau,[25] Schuerman,[26] Wilson and Ford,[27] and Turnbull and McIntosh.[24] The pontile nuclei were more affected than the white substance, except in one of the cases of Turnbull and McIntosh [24] in which the white subtance was more infiltrated than the gray. This is similar to the observations in my case, although the pons was not the area which showed the greatest infiltrations, but the centrum ovale. Also, contrary to the reported cases, the meninges, the medulla and the cervical cord were not infiltrated, while the infiltrations in the cerebellum were limited to the white substance around the dendate nucleus. The infiltrations in the meninges in all cases were slight, so that its absence in this case is not important. The upper cervical cord in one case of Turnbull and McIntosh was also not infiltrated, though infiltrated in the lower parts. In one of Perdrau's [25] cases, the whole cord was but slightly affected. Apparently the infiltrations in the cord vary from case to case. However, since in the cases of myelitis the medulla was also infiltrated, the inflammatory process in my case would seem to have stopped in the pons, not spreading to the cerebellum, the medulla or the cord.

The clear spaces around the blood vessels have been interpreted as a myelin sheath degeneration. It does not seem possible that the myelin sheaths could degenerate to such an extent as to be noticeable with the Spielmeyer stain without the degeneration producing fat globules in the affected areas. Fat globules are found only in the adventitia of noninfiltrated vessels near the cortex. The amount

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24. Turnbull, H. M., and McIntosh, J.: Brit. J. Exper. Path. **1**:89, 1920.

25. Perdrau, J. R.: J. Path. & Bact. **31**:17, 1928.

26. Schuerman, P.: Beitr. z. path. Anat. u. z. allg. Path. **79**:357, 1928.

27. Wilson, R. E., and Ford, F. R.: Bull. Johns Hopkins Hosp. **40**:337 (June) 1927.

of fat in this region is not greater than it is in children's brains in toxic or febrile conditions. Perdrau [25] also commented on the scarcity of fat, which he attributed to rapid absorption. It seems more likely that the clear spaces around the vessels are due to a mesodermal reaction and that the myelin sheaths have been pushed aside rather than destroyed. In old cases of encephalitis such mesodermal overgrowth round the vessels is commonly found.

Schuerman [26] stressed the occurrence of a glia net around the infiltrated areas, especially in the pons. It is not possible to find such a glia net in the Holzer preparations, except around both infiltrated and noninfiltrated vessels of the pons.

SUMMARY

A case of encephalitis and influenzal bronchopneumonia occurring in a child twelve days after vaccination for variola has been described. Since influenzal encephalitis as an entity has not been established, the clinical diagnosis rested between a sporadic case of encephalitis epidemica and postvaccinal encephalitis. The histopathologic observations in the brain were not similar to those found in epidemic encephalitis, since there was no perivascular infiltration of the gray substance around the ventricles and sylvian aqueduct, except in the lateral areas of the thalamus and the caudate nucleus. The chief areas of perivascular infiltration were in the centrum ovale. The infiltrations in the basal ganglia, the pons and the cerebellum were slight. There were no infiltrations in the meninges, the cord or the medulla oblongata. In spite of some dissimilarity to the reported cases of postvaccinal encephalitis, the histopathology on the whole conforms to that of this disease.

PATHOGENESIS OF AMAUROTIC IDIOCY *

PROF. CHARLES SCHAFFER, BUDAPEST, HUNGARY

In 1925, I expressed an opinion in an article in the ARCHIVES concerning the general significance of the Tay-Sachs form of familial idiocy; [1] I concluded that this is a germ layer disease, marked by the anatomic triad of ectodermal selectivity, segment selectivity and system selectivity. This theory is opposed by the new observations of Bielschowsky [2] and his followers (H. Kufs, B. Sachs) who claim that amaurotic idiocy is nothing more than a partial manifestation of a mesodermic disease, Niemann-Pick's disease, which as a metabolic disease affects the reticuloendothelial system. As a result of this endogenous metabolic disease, a deposition of lipoid substances occurs in the spleen, liver, thymus, suprarenal medulla, pancreas, the endothelial cells of the blood vessels and heart muscle cells; further, one finds fat droplets in the peripheral nerves, the ganglion cells and the spinal ganglia. According to Pick,[3] there is an "oversupply" (Ueberangebot) of lipoid metabolic products. The primary metabolic disturbance is a constitutional, congenital and familial disease, which occurs mostly in females, especially of the eastern Jewish race.

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* Submitted for publication, April 28, 1930.

1. Schaffer, C.: General Significance of Tay-Sachs' Disease, Arch. Neurol. & Psychiat. **14**:731 (Dec.) 1925.

2. Bielschowsky, M.: Amaurotische Idiotie und lipoidzellige Splenohepatomegalie, J. f. Psychol. u. Neurol. **36**:103, 1928.

3. Pick, L.: Der Morbus Gaucher und die ihm ähnlichen Erkrankungen, Ergebn. d. inn. Med., 1926, vol. 29.

Bielschowsky's view was determined by two features: the chemical identity of the degeneration products in both the Niemann-Pick and the Tay-Sachs disease, and the apparently not infrequent combination of the two diseases. It may be emphasized at the outset that neither the chemical identity nor the simultaneous occurrence of both diseases in one person proves the identity of the two conditions. Further, I would state that if the Tay-Sachs disease were a manifestation of the Niemann-Pick disease, it would follow that: (1) The disturbance of the reticuloendothelial system must be present in every case of Tay-Sachs' disease, as it would then be fundamental for this disease. (2) The occurrence of Tay-Sachs' disease alone without Niemann-Pick's disease would be impossible, for the tremendous overloading with lipoids which would occur in the Tay-Sachs disease as a manifestation of the mesodermal metabolic disturbance would be inconceivable without the primary disease of the metabolic mesodermal apparatus.

Before I state my opinion on this disputed question as to the identity of the pathogenesis of Niemann-Pick's and Tay-Sachs' disease, I shall refer especially to the fundamental picture of the degeneration in Tay-Sachs' disease, for in the interpretation of the changes of the ganglion cells I have come to a conclusion entirely different from that of Bielschowsky and Spielmeyer. In the opinion of these authors the pangangliocellular swelling in Tay-Sachs' disease results merely from the deposition of granules, which, according to Spielmeyer,[4] are demonstrable with the ordinary fat stains. In contradiction to this opinion, from a study of my specimens I came to the conclusion, as early as 1922 (therefore, before Marinesco,[5] in 1927), that the primary manifestation is not the formation of granules, but the volume increase of the hyalocytoplasm as a result of colloidal swelling of the latter. The primary phase of swelling is followed by the phase of precipitation, which develops gradually in such a manner that a few small granules appear in the swollen hyaloplasm and then gradually increase in number and in size. Finally the entire body of the ganglion cell comes to be packed with coarse, polygonal granules, the characteristic feature of which is that they stain intensively with Weigert's hematoxylin and therefore represent a lecithin-like substance. This morphologic phenomenon is demonstrated in figure 1, in which four swollen nerve cells can be seen. Cell *a* shows the beginning of the intracellular process; one sees the balloon-shaped swelling of a pyramidal cell with scattered fine granules in the protoplasm. In cell *b* the granules are already larger and fill completely the cell body in which the round granules, equal in size, are saturated with the stain. In cell *c* the increase in size of the granules is striking. Finally, in cell *d* the swelling and the precipitation process have reached their high point, for the hematoxylin granules are coarser and more polygonal and have accommodated themselves to the contours of the dilated intracellular network. It is my opinion that these cellular pictures, appearing side by side in one visual field, demonstrate emphatically the successive development of the pancellular swelling, from which it is evident that the deposition is not the primary and important factor, but the swelling, from which the precipitation of the intracellular granules gradually develops. Other interpretations of this series of pictures are less justifiable, unless one perverts the facts that present themselves to the eye of the observer.

4. Spielmeyer, W.: Vom Wesen des anatomischen Prozesses bei der familiären amaurotischen Idiotie, J. f. Psychol. u. Neurol. **38**:120, 1929.

5. Marinesco, G.: Nouvelle recherches sur la forme de Spielmeyer-Vogt de l'idiotie amaurotique et son mécanisme biochemique, Encéphale **22**:605, 1927.

In my demonstration of the ganglion cell process in Tay-Sachs' disease, as a histopathologist I confined myself to morphologic phenomena and considered the chemical side in my various articles only so far as to mention a prelipoid substance on the basis of the staining properties of the precipitation products. This prelipoid substance has a tendency toward affinity for the sudan stain, as Hassin [6] especially

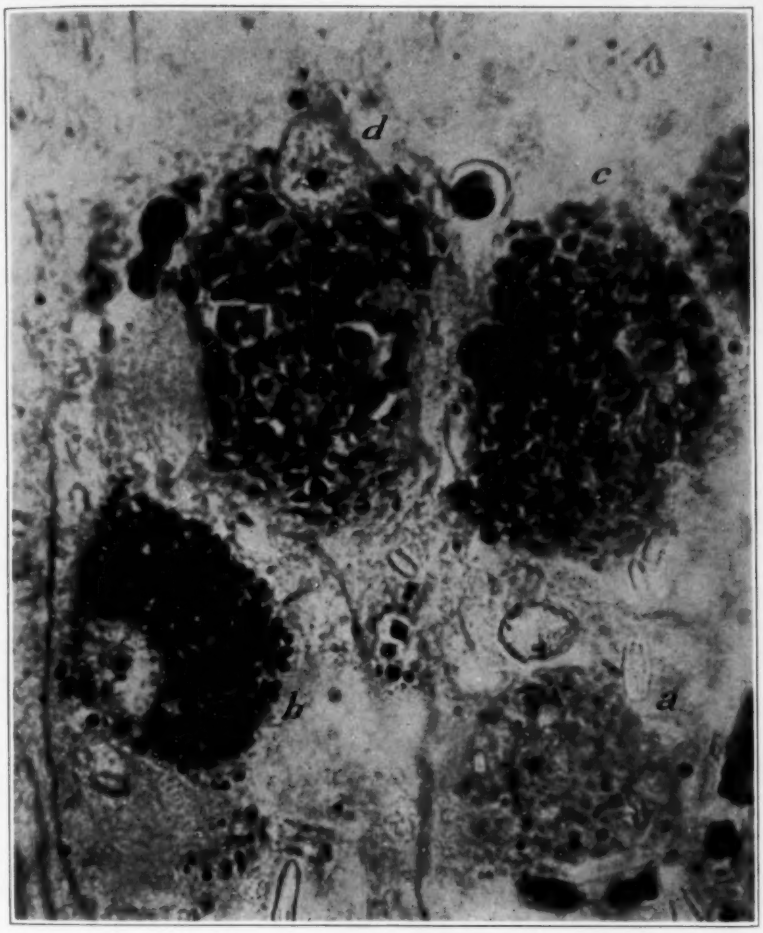

Fig. 1.—Oil-immersion photomicrograph from the frontal lobe in a case of amaurotic idiocy. Weigert's hematoxylin stain with Schaffer's modification (Arch. f. Psychiat. u. Neurol., 1922, vol. 64). *a* indicates a swollen nerve cell with initial precipitation; *b* and *c*, advanced precipitation; *d*, final stage of precipitation.

was able to demonstrate. These staining properties suggest a process that tends toward fatty changes. Marinesco assumed that by the splitting of the lipoproteins

6. Hassin, G. B.: A Study of the Histopathology of Amaurotic Family Idiocy (Infantile Type of Tay-Sachs), Arch. Neurol. & Psychiat. **12**:640 (Dec.) 1924.

hydrophil substances are produced, which he considers as phosphorus-containing lipoids. This splitting, in consequence of an increase of the molecules, causes an endosmotic current in the cell body. At the same time an oxidation disturbance occurs, which accounts for the depletion of oxydases in the swollen nerve cells.

The first question is: What is the origin of the prelipoid degeneration products of the ganglion cells? My opinion, which is based on anatomic observations, has always been that they arise locally; in other words, that they are granules which

Fig. 2.—From the cerebellum in a case of amaurotic idiocy. Cajal's cerebellar impregnation. *X* indicates a swelling of the cell body and initial dendrites; *XX*, "goose-feet."

are closely bound to the nerve substance, and consequently that they are ectodermal degeneration products. In opposition to this view, which has its origin in histopathologic observations, is the theory of Bielschowsky, discussed in detail previously, according to which the storing of lipoid masses in the central nervous system depends on an endogenous mesodermal disease of the reticulo-endothelial metabolic apparatus. As I have mentioned, however, if this were so, one should

find simultaneously in every case of Tay-Sachs' disease the fundamental changes that occur in the Niemann-Pick disease. Nevertheless, Strauss,[7] in numerous cases of Tay-Sachs' disease, failed to find the spleen and liver "pathologically enlarged." I believe that it is of foremost importance in this disputed question to compare the changes in the central nervous system in pure Niemann-Pick's disease with those in pure Tay-Sachs' disease, as Hassin [8] has already done. He found slight swelling of the dendrites and formation of vacuoles in some of the ganglion cells in

Fig. 3.—From the cerebellum in the case of Niemann-Pick's disease described in the text. Cajal's cerebellar impregnation.

Niemann-Pick's disease, and did not believe that the conditions are the same in the two diseases.

7. Strauss, I.: Discussion on the lecture of Sachs, B.: Amaurotic Family Idiocy and General Lipoid Degeneration, Tr. Am. Neurol. A., 1928.

8. Hassin, G. B.: Amaurotic Family Idiocy: Clinical and Pathologic Studies, Am. J. Psychiat. **8**:969, 1929.

I herewith present a case which brings out this comparison, a pure, histopathologically clear case of Niemann-Pick's disease, which Professor von Meyenburg of Zürich placed at my disposal.

REPORT OF CASE

The case was that of a boy, aged 1 year, the pathologico-anatomic diagnosis of which condition was "lipoid-cellular splenomegaly of the type of Niemann-Pick."

Fig. 4.—Cerebellum from a case of Tay-Sachs' disease. Weigert's hematoxylin stain (Schaffer's modification). Fuchsin contrast stain.

I received for study pieces of the cerebrum and cerebellum. For the purpose of comparison I have chosen the cerebellum because of its characteristic and impressive histopathologic structure.

I employed first the fibril impregnation method of Cajal. Figure 2 shows the cerebellum in Tay-Sachs' disease; figure 3, that in Niemann-Pick's disease. In the cerebellum from the specimen of Niemann-Pick's disease there is visible a certain degree of swelling of the Purkinje neurons of the cell body as well as of

the dendrites, especially at the points of bifurcation. The phenomenon of swelling is, however, without doubt more intensive in the specimens from Tay-Sachs' disease. I should say even that it occurs in an exaggerated form. The Purkinje cell bodies are more than twice the size of those in the specimen of Niemann-Pick's disease. Furthermore, one sees in Tay-Sachs' disease the characteristic plump swelling of the cell body together with the initial dendrites (fig. 2*X*). Finally, one notes the grotesquely exorbitant, well defined and sharply localized

Fig. 5.—Cerebellar gyrus from a case of Niemann-Pick's disease. Weigert's hematoxylin stain (Schaffer's modification). Fuchsin contrast stain. *X* indicates a bundle of swollen medullary fibers, *XX*, a necrotic area of the stratum granulosum. Especially noteworthy is the two or three-rowed layer of fat bodies in the area of Purkinje cells; further, the bordering black line on the cortical surface, representing the lipoid containing mesoderm (leptomeninges and blood vessels).

swellings of the dendrites, which Lewin[9] has aptly called "patitas de ganso" (goose-feet) (fig. 2*XX*). To summarize, it may be said that whereas swelling is

9. Lewin, S.: Sobre la idiocia amaurotica de los niños judíos de la Rusia blanca, Arch. argent. de neurol. **5**:66, 1930.

present in cases of Niemann-Pick's disease, it is nevertheless far less pronounced than in Tay-Sachs' disease.

The truly decisive difference appears, however, in Weigert's hematoxylin specimens. Figure 4 illustrates a cerebellar gyrus in Tay-Sachs' disease, in which it is evident that all of the cerebellar neurons are filled with hematoxylinophilic, prelipoid granules. This is strikingly demonstrable in the Purkinje neurons as well as in the cells of the stratum granulosum, as a result of which a well marked dark line appears between the molecularis and the white matter. The Purkinje neurons present themselves in a row of bottle-shaped bodies; in the stratum granulosum a more dustlike granulation is to be seen, because the cell body of the granule cells is much smaller. One also sees, however, diffuse finer granules in the molecularis, which represent the cell bodies of the star cells and basket cells.

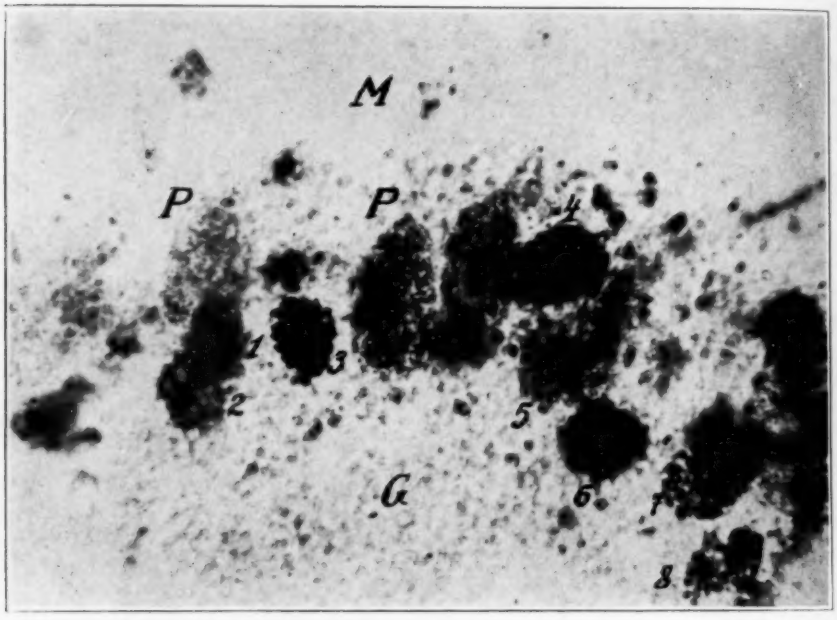

Fig. 6.—Cerebellar cortex from a case of Niemann-Pick's disease, same stain as in figure 5. Note particularly the structure of the fat bodies (*1* to *8*), which appear as coarsely granulated foam cells (lipoid transportation elements of mesodermal origin); the long oval Purkinje cells, *P*, delicately granulated; *G*, stratum granulosum, not containing lipoid.

The Weigert hematoxylin specimens of the cerebellum in Nieman-Pick's disease show an entirely different picture (fig. 5). The most marked observation appears at the border between the Purkinje neurons and the granulosum in the form of a layer of two or three rows of deep black spherical bodies, which are somewhat smaller than the swollen Purkinje cell bodies; they occur also scattered in the granulosum, but here are of a somewhat smaller size. One may say that these spherical bodies, which show a coarse granulation, increase in size the closer they lie to the Purkinje cell layer; it looks as if they had poured out of the white

matter, although they cease at the line of the Purkinje elements, for the molecularis is completely free from these spherical bodies. These are the well known "form cells" or "prelipoid bodies" of Niemann-Pick's disease (fig. 7). A notable fact is that degenerated medullary rays are to be seen in groups in the granularis; bundles of swollen medullary fibers are seen (fig. 5 *X*), which have a tendency to break down and to necrose (fig. 5 *XX*). I would emphasize especially that in Niemann-Pick's disease the Purkinje neurons alone possess a fine prelipoid granulation, while the nerve cells of the molecularis and of the granulosum remain completely free from this; in consequence, a marked clearing appears in the granulosum, producing a striking difference from the picture of Weigert specimens in Tay-Sachs' disease.

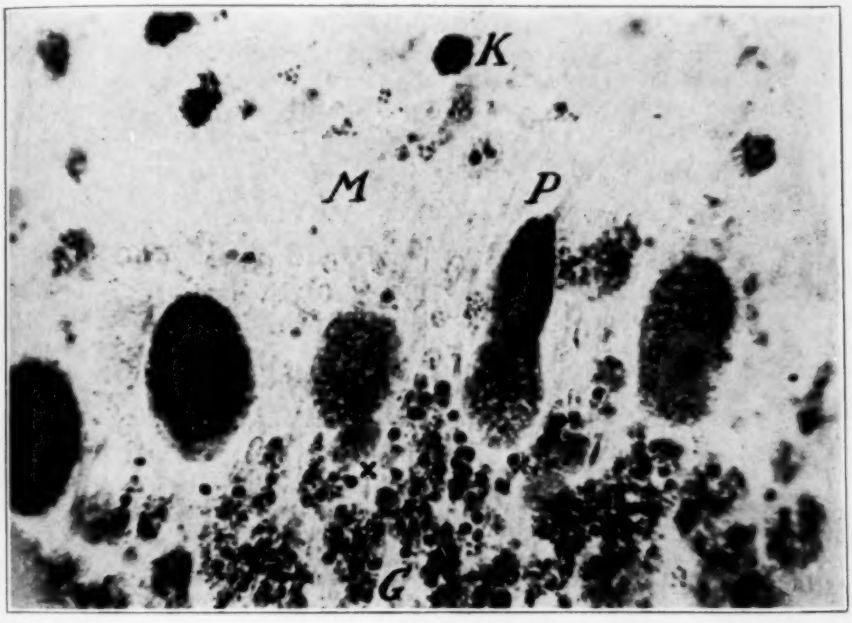

Fig. 7.—Cerebellar cortex from a case of Tay-Sachs' disease, same stain as in figure 5. Note the granulated and swollen Purkinje cells, *P*, below which the fine lipoid-containing stratum granulosum, *G*, without fat bodies appears; *X* represents a gliogenous transportation element; *M*, stratum moleculare in which basket cells containing lipoid granules are visible. Oil-immersion photomicrograph.

The conditions described are shown in figures 6 and 7 under an oil-immersion lens. Figure 6 represents the cerebellum in Niemann-Pick's disease and shows four swollen Purkinje cells filled with fine hematoxylinophilic granules. Among these cells appear the coarsely granulated "fat bodies" or "foam cells" (fig. 6, *1* to *8*); figure 7, from a case of Tay-Sachs' disease, shows five Purkinje cells, which are conspicuous by their swelling as well as by their coarse prelipoid granulation. Not a single fat body is visible; however, mobile, so-called transport cells, apparently of glial origin (fig. 7 *X*), are seen, in which prelipoid granules cannot be found.

COMMENT

From this short description it is obvious that fat bodies characteristic of Niemann-Pick's disease, which are the mesodermal transport elements of lipoid metabolic products, are entirely absent in pure cases of Tay-Sachs' disease; from this one must conclude that in pure Tay-Sachs' disease the histopathologic evidence of a lipoidal metabolic disease is wanting. To this it may be added, as an important fact, that the changes in the vascular system are essentially different in the two diseases. In Niemann-Pick's disease, dense, fine, dustlike granules are scattered in the leptomeninges and in the vascular intima, as can readily be seen in figure 5, as a black lineal contour covering the cortical surface. In Tay-Sachs' disease the meninges and vascular system are absolutely free from this granulation.

To summarize, one may say that the prelipoid picture of Tay-Sachs' is essentially different from that of Niemann-Pick's disease. In Niemann-Pick's disease the fat-storing cells appear in the nerve parenchyma as prelipoid bodies; further, the mesoderm (leptomeninges and vascular system) shows a pronounced fine granulation with lipoid substances. In Tay-Sachs' disease, on the contrary, the fat-storing cells are completely absent, and the mesoderm is indifferent; in other words, it is free from granulation. From this behavior it is clear that in Niemann-Pick's disease the mesoderm and in Tay-Sachs' disease the ectoderm is primarily diseased; one is therefore dealing with diseases of derivates of two different germ layers. One should therefore not identify Tay-Sachs' disease with Niemann-Pick's disease on the basis of similar chemical staining properties of the degeneration products, as has already been emphasized. Finally, it is not surprising that the central nervous system, which is rich in myelin substances, should give rise to lipoid substances in its breakdown. Lipoids appear as products of metabolic disturbance in Niemann-Pick's disease also, though here in the characteristic foam cells. The lipoid in Niemann-Pick's disease saturates the whole organism and therefore also the central nervous system. One should not be surprised, therefore, at the presence of lipoids in the central nervous system, but should be careful not to assume from the mere presence of such substances in the central nervous system a metabolic disturbance having its origin in the reticuloendothelial system; on the other hand, it is erroneous to make a diagnosis of Tay-Sachs' disease from the appearance of lipoids in the central nervous system in cases of Niemann-Pick's disease. The latter point has been especially well illustrated by Smetana,[10] who found a classic case of Niemann-Pick's disease without evidence of Tay-Sachs' disease in a Jewish girl, aged 20 months.

Niemann-Pick's disease should be differentiated from Tay-Sachs' disease in the sense that the former is an endogenous mesodermal and the latter an endogenous ectodermal disease; both are constitutional, congenital and familial diseases, characterized by racial predisposition. On the basis of the germ layer autonomy, they are two different diseases which, however, may occur simultaneously on account of their endogenous basis; in this case one is dealing with a combined ectomesodermal disease, that is, a combined germ layer disease. In the combined form, however, the disease of the central nervous system is not a partial manifestation of the endogenous mesodermal disease, as Bielschowsky and Kufs[11]

10. Smetana, H.: Ein Fall von Niemann-Pickscher Erkrankung, Virchows Arch. f. path. Anat. **274**:697, 1930.

11. Kufs, H.: Ueber einen Fall von Spätform der amaurotischen Idiotie mit atypischem Verlauf und mit terminalen schweren Störungen des Fettstoffwechsels im Gesamtorganismus, Ztschr. f. d. ges. Neurol. u. Psychiat. **122**:395, 1929.

believe, but is a genuine ectodermal process, as is well seen from the fact that amaurotic idiocy can appear independently of the Niemann-Pick disease. A sharp boundary line should be drawn between the endogenous mesodermal and the endogenous ectodermal disease; both represent the inherent weakness—"abiogenesis" or "abiotrophy"—of one germ layer. I have come to the conclusion that amaurotic idiocy occurs only when endogenous weakness of the ectoderm is inherent. Naturally, this definition does not exclude the possibility that the mesoderm as well as the ectoderm may be congenitally weak in the same person; in this case a Niemann-Pick plus a Tay-Sachs disease may develop, as was realized in the case of Hamburger,[12] a striking example of combined disease of the germ layer. Such germ layer combinations are well known in pathology; for example, mesodermal muscular dystrophy with ectodermal spastic spinal paralysis, or bone dystrophy with familial spinal amyotrophia, either combined in one person or the parent suffering from one endogenous disease and the child from the other. In such cases the heredofamilial character stands in the foreground as a stigma of the endogenous feature of the disease.

12. Hamburger, R.: Lipoidzellige Splenohepatomegalie (Typus Niemann-Pick) in Verbindung mit amaurotischer Idiotie bei einem 14 Monate alten Mädchen, Jahrb. f. Kinderh. **116**:41, 1927.

HEREDITARY OPTIC ATROPHY (LEBER'S DISEASE)*

H. Houston Merritt, M.D., Boston

Hereditary optic atrophy, or Leber's disease, is an unusual condition, and has received little attention in American medical literature. Moreover, it is of interest to neurologists because of its close relationship to other heredodegenerative diseases of the central nervous system. Therefore, it was thought advisable to report in detail a case of this disease that was observed recently at the neurologic service of the Boston City Hospital, together with the family history of the patient and a brief review of the literature on the subject. Following is a summary of the records of this patient from examinations made at the Boston City Hospital on Aug. 5, 1929, by Dr. Stanley Cobb and his staff, and at the Wilmer Institute of Johns Hopkins Hospital on Nov. 25, 1929, by Dr. William H. Wilmer and his staff. The patient was referred to Dr. Cobb and Dr. Wilmer by Dr. John M. Wheeler of New York, who has kindly given permission for this report.

REPORT OF CASE

History.—A man, aged 27, had been working as a clerk in a stock and bond firm. His chief complaint was of loss of vision of one year's duration. He stated that there had been a history of eye trouble in his family for three generations. One brother of the patient's mother had the same trouble as the patient, with the onset at the age of 30. Another brother of the mother died at the age of 34, and had had trouble with his eyes for a few years previous to death. A first cousin of the mother also had the same trouble. The patient's father and

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* From the Department of Neuropathology, Harvard Medical School and the Neurological Service of the Boston City Hospital.

mother had come to the United States from Switzerland about forty years before this case was observed, and a detailed history of the family was not obtainable. The uncle of the patient who had similar eye trouble also emigrated to this country. His children had had no visual troubles. The uncle was examined by Dr. John M. Wheeler, of New York, and the condition of the eyes was described as similar to that of the patient. The patient's mother had no trouble with her eyes, but the patient said that she had a highly arched foot similar to his own. The patient's father had webbing of the toes. The father and mother were not related, and no history of nervous, mental or eye diseases in the family of the patient's father was obtained.

The history was without significance until the onset of the present illness. There was no history of alcoholism or excessive smoking. The patient had had measles, mumps and jaundice as a child. He had not been exposed to industrial hazards and there was no history of the taking of drugs. He had been troubled with frontal headaches for a number of years, but these were not severe and did not interfere with work. In August, 1928, he first noticed a blurring of vision in the left eye. This progressed rapidly for a few weeks, and then remained stationary. He consulted various physicians, and in an endeavor to remove all possible foci

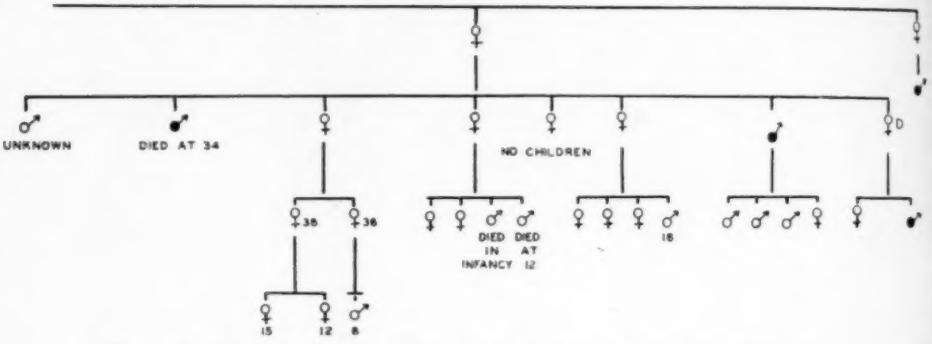

Fig. 1.—The family history of the patient as charted according to the severity of the disease. ♂ indicates the normal male in the family; ♀, the normal female, and ●♂, the male with visual loss.

of infection, all sinuses were explored in October, 1928, and the tonsils were removed. One suspicious tooth was extracted. Vision remained good in the right eye, and the patient was able to continue work, which required considerable use of the eyes, until May, 1929, when the right eye suddenly began to fail. The visual loss in the right eye progressed for approximately four weeks, and the patient was unable to continue work. He could not read, but he was able to get around the home and the streets unaided. He recognized the form of objects, but was unable to distinguish people until he recognized their voices. With the onset of the failure of vision, the headaches ceased.

Physical Examination.—The following observations were made: the blood pressure was slightly low, 100 systolic and 55 diastolic; the feet had an unusually high arch, similar to the clubfeet seen in Friedreich's ataxia, and there was webbing of the toes of both feet.

Neurologic Examination.—The cranial nerves were normal, except for the optic nerve, the condition of which will be described, and the presence of slight strabismus, which was thought to be due to the visual defect.

The motor system was normal throughout, and no disturbance in cutaneous, muscle, joint or vibratory sensations was detected. The deep reflexes in the arms (biceps, triceps and radial periosteals) and in the legs (quadriceps and achilles) were normally active. The abdominal and cremasteric reflexes were normal, and stimulation of the plantar surface of the foot gave plantar flexion of the toes. There was no Romberg sign.

Ophthalmologic Examination.—On Aug. 5, 1929, the external condition of the eyes was normal. The conjunctivae were clear—not injected. The corneas and lenses were normal. Both pupils measured 3.5 mm. in diameter. They reacted to light a little sluggishly. Visual acuity, corrected, was: right eye 2/60, left 1/60. The patient recognized all the primary colors with the exception of green, which he mistook for gray. He recognized candle motion at 20 feet. The visual fields, taken with a tangent screen, showed: a central scotoma for white and colors, with a normal peripheral field in the left eye and a paracentral scotoma with normal peripheral fields in the right eye. The visual fields taken with a stereocampimeter showed a central scotoma of the left eye and a paracentral scotoma of the right eye.

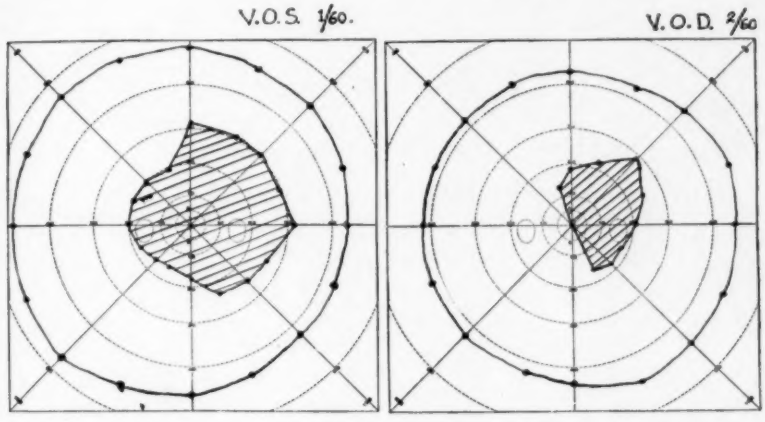

Fig. 2.—The visual fields of the patient as they appeared on Aug. 5, 1929. The fields were taken on a tangent screen, 5 mm. white disk, with the patient 1.5 meters from the screen.

Ophthalmoscopic Examination.—In the left eye, the media were clear. The disk was about normal in size, and the margins were clearly outlined. There was a marked whitening of the papillomacular bundle with some pallor of the whole temporal side of the disk. The nasal side of the disk had a more normal color. The vessels were normal in size and were not tortuous. There was a slight amount of streaking of the retina around the vessels near the edge of the disk. In the right eye, the changes in the nerve seemed to be more recent. The upper and nasal borders of the disk were slightly blurred, and there was a slight edema of the surrounding retina. The temporal half of the disk was definitely pale. The vessels were similar to those on the left. The periphery of the retinas was normal.

When the patient was examined by Dr. Wilmer on Nov. 25, 1929, the condition of the eyes was similar to that already described, except that there had been a subsidence of the inflammatory process in the right eye. Visual acuity, uncorrected, was found to be: right eye, 2/100; left eye, 3/70; corrected, it was right eye, 2/70; left eye, 4/70. The visual fields taken at this time with an arc perim-

eter were essentially the same as those taken on Aug. 5, 1929, except that the scotoma in the right eye was somewhat more centrally located.

Laboratory Examinations.—Examination revealed: red blood corpuscles, 4,250,000 per cubic millimeter; hemoglobin, 85 per cent; white blood corpuscles, 6,550; the blood smear, normal. The urine was clear, of neutral reaction and

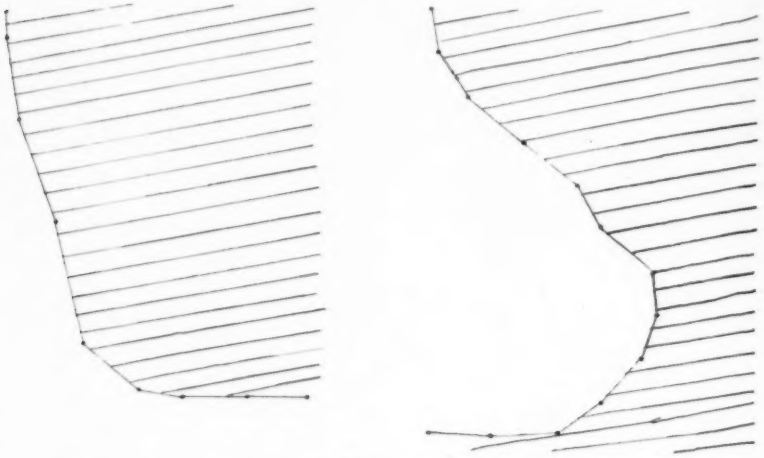

Fig. 3.—Stereocampimeter fields as they appeared on Aug. 5, 1929.

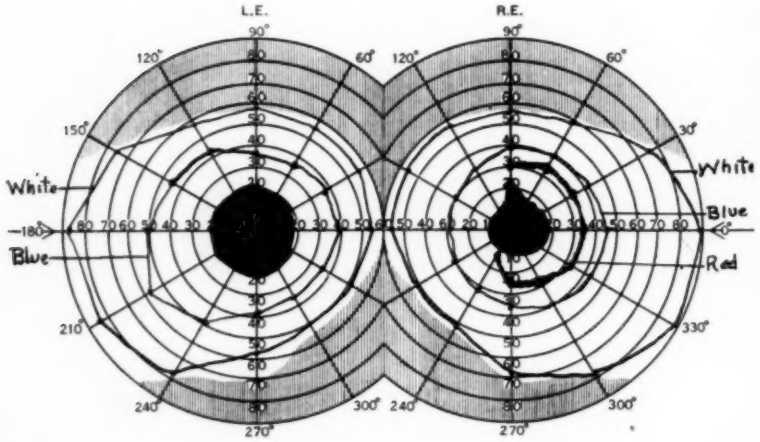

Fig. 4.—The visual fields of the patient as they appeared on Sept. 25, 1929. These fields for form and color were taken with an arc perimeter.

had a specific gravity of 1.015; albumin and sugar tests, microscopic examination of the sediment and the Kahn test of the blood were negative.

Lumbar Puncture.—The cerebrospinal fluid pressure was 130 mm. of water, and the dynamics of the system were normal. The fluid was clear and colorless, and as a result of a chemical examination, Dr. Fremont-Smith's laboratory reported

the total protein as 0.031 per cent and the colloidal gold as 0011100000. The Wassermann reaction of the fluid was negative.

Dextrose Tolerance Test.—The blood sugar level during fasting was 84 mg. per hundred cubic centimeters, with a prompt rise to 110 mg. on the ingestion of 100 Gm. of dextrose and a fall to normal in ninety minutes.

Roentgenographic Studies.—The skull showed no evidence of pressure; the sella turcica was normal in size and shape. The lungs were normal.

COMMENT

Hereditary optic atrophy was first described by Leber [1] in 1871, although to von Graefe [2] should be given the credit for being the first to report the condition. Von Graefe observed three cases in one family in 1858. Leber studied this family and reported a total of fifteen cases in four different families. The description of the disease by Leber, and his statements as to the mode of onset, the influence of heredity and the prognosis are classic. Little has been added to the knowledge of the disease by subsequent authors, except that minor variations of the clinical pictures are described.

After Leber's description, the disease received much attention in the literature, particularly at the beginning of the present century. Hormuth,[3] in 1902, in a very thorough study, collected from the literature and his personal observations, over 300 cases in 80 different families. He analyzed these cases carefully. In 1909, Nettleship [4] chose as the subject of the Bowman's Lecture, "Some Hereditary Diseases of the Eye," in which he collected 547 cases of Leber's disease, published and unpublished.

James Taylor and Gordon Holmes [5] reported eleven cases in two families and called attention to other neurologic abnormalities in some of the patients. Ferguson and Critchley,[6] in 1928, reported four cases of the disease in one family in which other neurologic changes were found; they called attention to the relationship of the disease to the heredofamilial ataxias. Other recent studies of the disease have been made by Hogg,[7] Wilmer,[8] Reeder [9] and Mann.[10] From a review

1. Leber: Ueber hereditäre und congenital angelegte Sehnervenleiden, Arch. f. Ophth. **17**:249, 1871.

2. Von Graefe: Ein ungewöhnlicher Fall von hereditäre Amaurose, Arch. f. Ophth. **4**:266, 1858.

3. Hormuth. Beiträge zur Lehre von den hereditären Sehnervenleiden, Beitr. z. Augenh. **5**:63, 1902.

4. Nettleship: Bowman Lecture: On Some Hereditary Diseases of the Eye, Tr. Ophth. Soc. U. Kingdom **39**:57, 1909.

5. Taylor, James; and Holmes, Gordon: Two Families with Several Members in Each Suffering from Optic Atrophy, Tr. Ophth. Soc. U. Kingdom **33**:95 and 116, 1913.

6. Ferguson, F. R., and Critchley, M.: Leber's Optic Atrophy and Its Relationship with the Heredo-Familial Ataxias, J. Neurol. & Psychopath. **9**:120, 1928.

7. Hogg, G. H.: Hereditary Optic Atrophy, M. J. Australia **1**:372 (March 24) 1928.

8. Wilmer, W. H.: Hereditary Factors Responsible for Development of Optic Atrophy and Retinitis Pigmentosa, Arch. Neurol. & Psychiat. **12**:137 (Aug.) 1924.

9. Reeder, J. E.: Hereditary Optic Atrophy, Am. J. Ophth. **10**:429 (June) 1927.

10. Mann, I. C.: Retinitis Pigmentosa and Leber's Disease, Ann. Eugenics **3**:77 (April) 1928.

of the literature, chiefly from the facts brought out by Hormuth [3] and Nettleship,[4] the following facts are found to be characteristic of the disease:

Etiology.—The disease is always inherited. Syphilis and tuberculosis play no part in the cause, but it is thought that acute infections and other diseases may precede the onset. Hormuth's theory is that affected persons are born with a weak papillomacular bundle, and the factor precipitating the onset of the disease is entirely unknown.

The disease generally affects more than one member in a family, and can be traced for several generations in a line, although it usually skips one or more generations. Males are chiefly affected in the ratio of 300 males to 60 females in Nettleship's statistics, and it is transmitted almost entirely by females; he was able to find only 6 cases in the literature in which it was transmitted by males, and 4 of these were in one family. Consanguinity plays no part in the transmission. According to Blegvad and Rönne,[11] there are occasionally cases in which there is no family history of the disease. Every familial disease, however, must have a starting point, a first case.

Onset.—In the large majority of patients in Hormuth's and Nettleship's series the onset occurred between the ages of 18 and 30, with 6 and 52 years as the extremes; they both state that there is a tendency toward anticipation in successive generations. The onset is usually sudden in both eyes, and is described as a mist over the eyes, but in some cases months may separate the time of onset in the two eyes. The onset is often preceded by severe frontal headaches.

Progress.—The disease usually progresses to a certain stage in a few weeks and then remains stationary. It rarely progresses to complete blindness, and only a few cases of complete recovery are reported. However, there is a tendency for slight improvement in vision. This is explained by Dr. Wilmer as being due to the fact that the patient learns to accommodate himself to his central scotoma and to use the periphery of his retina to better advantage.

Character of the Visual Loss.—The visual loss is practically always a central (rarely paracentral) scotoma for white and colors or for colors alone, with normal peripheral fields. Contraction of the fields has been reported, but never a quadrantic loss.

In the early stages the fundus picture is that of an axial neuritis; later, it is one of postneuritic atrophy, involving chiefly the temporal fibers.

Concomitant Diseases.—Nettleship stated that the atrophy is rarely accompanied by other congenital diseases of the eye, but all authors agree that it is occasionally associated with epilepsy and hysteria. Pines and Tron [12] reported six cases of Leber's disease in one family. Three of these cases showed nystagmus, tremor of the hand and increased deep reflexes. Taylor and Holmes [5] collected several cases of Leber's disease in which there were in addition to the optic atrophy various neurologic abnormalities, such as absent knee or ankle jerks, dull aching pains, some anesthesia and analgesia, slight sphincteric disturbances and mental changes. Ferguson and Critchley [6] reported four cases in one family, one of which presented epileptic attacks and mental defect, and another, signs of pyramidal

11. Blegvad and Rönne: Ueber die Klinik und Systematik der Retrobulbärneuritiden, Klin. Monatsbl. f. Augenh. **65**:206, 1920.

12. Pines and Tron: Hereditäre Neuritis optica (Lebersche Sehnervenatrophie), Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:763, 1925.

disease, ataxia and sensory loss. These authors reviewed all cases in the literature in which neurologic abnormalities were reported. They quoted Leber's statement that in many of the families minor neurologic abnormalities occurred, and that the affected or unaffected members occasionally suffered from periodic headaches, migraine, vomiting and giddiness and were often nervous. They also called attention to the nine families in Nettleship's series in which epilepisy was also present and to the reported cases of Marie's ataxia in which there was an accompanying optic atrophy, and mentioned that optic atrophy also occurred occasionally in Friedreich's ataxia. Their cases and the review of the literature, in their opinion, supported Raymond's unitary conception of the heredofamilial diseases of the nervous system. They concluded that Leber's disease is a "neuronic abiotrophy, limited, in typical cases, to the optic nerve."

In this connection it is interesting to note that our patient had club feet similar to those seen in Friedreich's ataxia, which apparently were inherited from his mother.

Differential Diagnosis.—The diagnosis is generally not difficult to make when the typical family history and visual disturbance (central scotoma with normal peripheral fields) are found. It is to be distinguished from the tobacco-alcohol amblyopias by the difference in the patient's age at the onset of the two conditions, the latter generally coming on late in life and improving with abstinence from tobacco and alcohol. Retrobulbar neuritis occurring in more than one member of a family might offer difficulty. Tabetic optic atrophy is readily recognized by its progressive course and the presence of other signs of syphilis.

Therapy.—No therapy has been of any value. The chief point is that the disease generally remains stationary after reaching a certain point, and the patient should be encouraged to adjust his life to the amount of vision preserved.

UNIQUE POSTENCEPHALITIC SYNDROME: COMPLETE PUPILLARY IMMOBILITY, HIRSCHSPRUNG COLON AND VESICAL INCONTINENCE*

D. M. Olkon, M.D., and Hallard Beard, M.D., Chicago

Complete pupillary inaction on both sides in a young person without apparent involvement of the classic areas of the brain, cord, cranial nerves and sympathetic system is rare; hence this report.

REPORT OF CASE

Clinical History.—R. M., of Italian parentage, aged 11, was fairly well developed, without apparent physical abnormalities and generally well. He was admitted to the pediatric department of the Research and Educational Hospitals

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* From the Departments of Neurology and Ophthalmology, University of Illinois College of Medicine and Research and Educational Hospitals. Case presented before the Clinical Conference, College of Medicine, University of Illinois, March 6, 1930.

on Sept. 16, 1929, on account of a chronic obstipation with vesical incontinence, which had existed from the earliest childhood. A diagnosis of chronic Hirschsprung's disease was made. During consultation for the vesical incontinence, the rigidity of the pupils attracted attention.

Three years before, he had had convulsions for one day, which were attributed to constipation. He fully recovered, and had had none since. The general behavior of the boy was good, his intelligence and cooperation being very good. His intelligence quotient with the completion test was 112, although he had not attended school on account of the incontinence. The past history of the patient and his parents was of no importance. The parents said that they did not suffer from alcoholism or syphilis.

Examination.—The general ocular and palpebral muscles were active; the left pupil was smaller than the right, and both were rigid to light and in convergence. With very strong illumination, the left pupil showed a faint response, but this was soon lost on the second or third trial. Vision was good, and the fundi were normal. A feature that attracted attention was the markedly distended abdomen, with a palpable colon of 2½ inches (6.27 cm.) in diameter in the left side of the abdomen, a visible peristalsis of the transverse colon, and atonicity of the entire abdominal musculature.

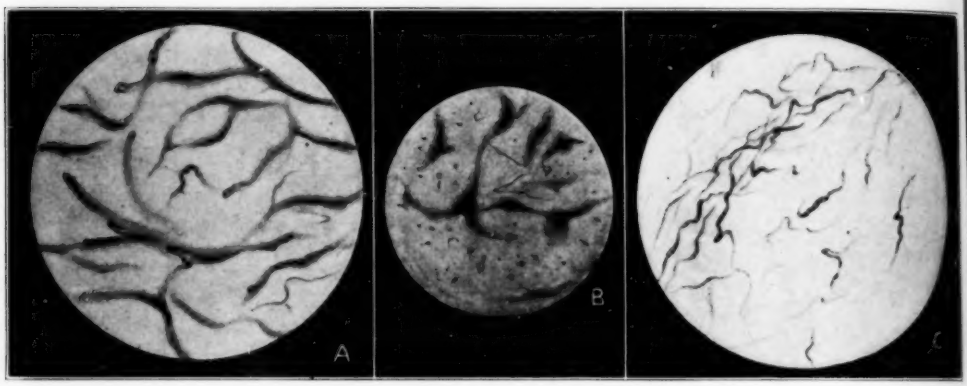

Fig. 1.—Three photomicrographs of capillary beds (after Muller). *A* shows the marked dilatation of the larger capillaries with many partly filled and deformed archicapillaries; *B*, the pronounced dilatation in atonic large capillaries, and irregular and sparse distribution of the comma-like archicapillaries; *C*, the prolonged atony of the subpapillary plexuses with spasm of the capillaries and the formation of confluent loops and tangles.

Neurologic Examination.—The external ocular movements were good and equal on the two sides. There was no involvement of the cranial nerves. All reflexes were present and equal on the two sides, and no pathologic reflexes were apparent. The boy's gait was normal, and coordination good. Spina bifida occulta and aperta were absent.

There was marked vasomotor instability with pallid dermographia, which slowly became diffusely red and remained for ten minutes and longer, and the Aschner sign was present. The patient also had a slight tremor of the tongue.

The surface capillaries showed marked atony, dilated subpapillary vessels, deformed and elongated archicapillaries and the formation of confluent loops indicative of active changes in the capillary system.

Roentgen Examination.—The long bones, head, sella and sinuses were normal.

A barium sulphate enema showed a very large colon with visible peristalsis through the abdominal wall. Cystoscopy of the urinary bladder gave negative results, except that the left ureter was dilated.

Serologic Examinations.—The Wassermann, Kahn and Lange reactions with the blood and spinal fluid and the cell and globulin estimations were all within normal limits. Examinations of the urine and feces gave normal results. Wassermann, Kahn and Lange tests with the blood and spinal fluid for syphilis were also made on the father, mother, an older sister and a younger brother. All reactions were negative.

Ophthalmologic Study (Dr. Beard).—A refraction under cycloplegia showed a moderate amount of hyperopia in both eyes, and a careful search of the eyegrounds failed to reveal any abnormality. The inequality and inactivity of the pupils, however, to both light and convergence, were definite. The right pupil was 4.5,

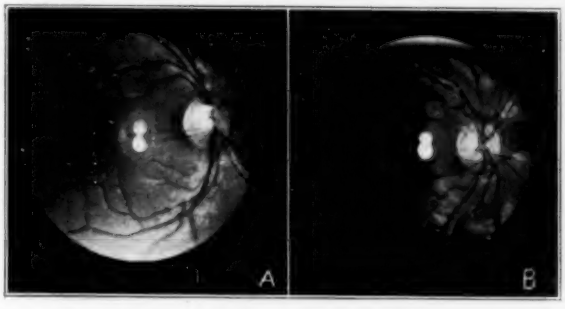

Fig. 2.—Photomicrographs of the fundus oculi taken with a Nordensen-Zeiss fundus camera. *A*, shows the normal fundus; *B*, the patient's fundus. The two white spots in the center of each photomicrograph are artefacts of illumination, but the patient's fundus is normal.

the left 3.5 mm. in diameter, and the strongest light produced a faint twitch only in the left. The question then arose as to whether there existed a break in the continuity of the pupillary reflex arc for light or whether there was a defect in the iris musculature itself, as suggested by the absence of the convergence response.

The presence of some sphincter tonus was established by the fact that when the eyes were atropinized, the pupils dilated to nearly 6 mm. Instillation of pilocarpine hydrochloride (after recovery from atropine mydriasis) produced contraction of the pupils to 1.5 mm., proving the integrity of the sphincter iridis and of its neuromuscular junctions. Owing to the patient's youth and illiteracy, it was difficult to measure his power of accommodation, but we were convinced that he could clearly distinguish fine print at reading distance.

Color vision and the fields for form and color were normal. Photographs of the fundi, taken with the Nordensen-Zeiss fundus camera, are shown in figure 2.

COMMENT

Clinical and laboratory observations have not given any clue to the possible cause of the rigid pupils. Syphilis was suspected, but the negative serologic, neurologic and developmental observations in the patient and the negative serologic observations in the parents and members of the immediate family speak against a syphilitic etiology.

With the exception of the retained accommodation, the results of these tests point to a central defect of innervation, and a consideration of the nerve pathways involved in the reflex arc would place the lesion most probably in the third nerve nucleus itself—in the group of cells governing pupillary contraction alone, possibly in the fibers proceeding from them. Only such a localization would be compatible with loss of convergence contraction, while the far more common Argyll Robertson reaction may be accounted for by lesions central to the third nerve nucleus.

The assumption of such a restricted area for the localization of a lesion capable of causing the conditions described may seem far-fetched, yet it is well to note that the power of convergence itself was unimpaired. The conception of an atony of the iris musculature or innervation may well be entertained here, as the dilatation of the pupils obtained by atropine was only moderate, not maximal (8 or 9 mm.), as would be expected in so young a subject. Moreover, the miosis obtained by pilocarpine hydrochloride was marked.

Cases of mydriasis and accommodation paralysis not due to syphilis have been reported by Oppenheim.[1] Pana [2] called attention to rigidity of the pupils caused by intestinal autointoxication, but he did not give a detailed history of the case. Oppenheim cited a case of tabes in which the only symptom for over ten years was that of rigid pupils, and another case of healed syphilis in which for fifteen years the rigid pupils remained the only symptom of the disease. Cases of rigid pupils following epidemic encephalitis have been reported by Wimmer [3] and others. There is a vast literature on the subject, but in all of the foregoing instances there was apparently some known specific factor.

In the case reported here neither the history nor the observations give a clue to the etiology. There are observations, however, which it is well to consider: the outstanding visceral atony, the Hirschsprung colon and the vesical incontinence point to a tonus disturbance of central origin. Significant also is the fact that the marked atony of the viscera and bladder, as well as the incontinence, came on early in the life of the patient, the incontinence in particular at a time when it was difficult to distinguish between the normal spontaneous bladder emptying of infancy and that of an incontinence of central origin. Is it not probable that Hirschsprung's disease, continuous incontinence and the general atony, which is, after all, a form of severe muscle atony, have a common etiology? May it not also be true that the rigidity of the pupils belongs to the same syndrome? This opinion is suggested for the following two reasons: 1. The pupillary rigidity does not seem to be a true paralysis but rather a severe tonus disturbance, as was demonstrated by the response of the sphincter pupillae to homatropine hydrobromide and pilocarpine hydrochloride. 2. Muscle atony of toxic origin may be extreme and yet not be true paralysis.

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1. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, ed. 7, Berlin, S. Karger, 1923.

2. Pana, cited by Oppenheim (footnote 1).

3. Wimmer, A.: Epidemic Encephalitis, London, William Heinemann, 1924.

The second reason is demonstrated in the following cases: A young man, aged 19, developed severe disturbances of phonation;[4] his speech resembled that of typical bulbar paralysis. On detailed examination there were apparent: no paralysis of any of the muscles of speech, no atrophy, no reaction of degeneration to electrical stimulation, no sensory disturbances and no bodily muscular disturbance. In a word, there was nothing peripherally that could explain the defect of speech. On referring back, however, there was a history of an illness three years before, which lasted three weeks, with definite lethargic symptoms. Now there is a beginning parkinsonism, tremor of the tongue, excessive sialorrhea, a positive Aschner sign, excessive hyperhidrosis and other vasomotor disturbances; these accord with the conception that these are encephalitic sequels, and the vocal myasthenia is doubtless one of the syndromes of the postencephalitic substrate.

Another recent case is also instructive. A man, aged 45, with a marked postencephalitic syndrome — slow gait, cogwheel rigidity of joints, masked expression, tremor of the tongue, spontaneous tremor of the hand and a clear history of having had epidemic encephalitis five years before — developed complete loss of ocular motility. The eyes remain fixed and staring, yet on electrical stimulation and quick effort, the eyes move; but to all appearances the eye muscles are fixed. This case likewise is not a paralysis in the true sense; there is an extreme atony with asynergia of the ocular muscles. Similar atony is found in postencephalitis in the muscles of expression of the face, in the cogwheel rigidity of the limbs and in the general slowness of bodily movements.

The case of an Italian boy, aged 11, presents a form of muscle atony that cannot be ascribed to myopathies, myasthenia or dystrophies, or to degeneration. The general picture suggests a possible inflammation of the brain during early infancy. That encephalitis can effect the entire cerebrospinal axis has been reported by Lisa[5] and others. In Lisa's case in particular, section showed hyperemia with punctate hemorrhages throughout the brain and cord.

May it not be that the marked sympathetic disturbances of the colon, bladder, small intestines and pupils all belong to a common etiology? All of them are under the influence of the cerebral centers as well as of local innervation. It is difficult to explain the modus operandi, but the pathology of the Argyll Robertson phenomenon in tabes also stands in need of explanation.

This case presents a difficulty of localizing the specific etiologic factor, but the specific centers of innervation involved and the kind of pathology present are obvious. Nevertheless, the disorder is highly suggestive of a midbrain, striatum and basal ganglia involvement; hence a diagnosis of encephalitis of early infancy would seem to be warranted from the present observations and from retrospect.

CONCLUSION

We are inclined to consider the rigidity of the pupils, Hirschsprung's colon, vesical incontinence, vasomotor imbalance and capillary changes as due to the same etiologic substrate, namely, encephalitis of early infancy.

310 South Michigan Avenue.

30 North Michigan Avenue.

4. Olkon, D. M.: Vocal Myasthenia of Postencephalitic Origin: Report of a Case, Arch. Neurol. & Psychiat. **24**:570 (Sept.) 1930.

5. Lisa, cited by Neustaedter: Clinical Neurology, Philadelphia, F. A. Davis Company, 1929.

THE USE OF THE SCANNELL BLOOD TRANSFUSION CHAMBER IN LUMBAR INJECTION OF AIR*

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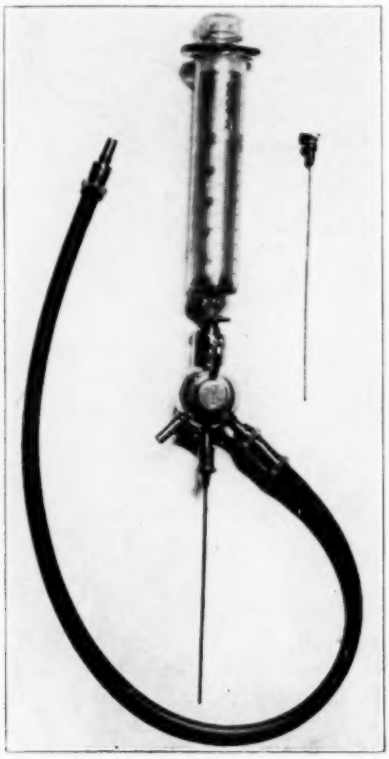
The number of indications for lumbar injection of air for both therapeutic and diagnostic reasons make the need for a simple technic of the operation apparent. Following is a description of a method that materially facilitates the procedure.

After the lumbar puncture has been performed, the Scannell blood transfusion chamber is attached to the lumbar puncture needle at the middle point of insertion, or the one which is normally used for the salt solution. A Luer adaptor is placed on the opposite side, so that a 10 cc. Luer syringe may be used. A rubber tube is attached to the left point of insertion to drain away the spinal fluid. Over the right outlet, a piece of sterile gauze is attached by means of a rubber band; this is in order to strain the air through a sterile substance.



Scannell blood transfusion chamber.

At any time during the operation, the syringe may be withdrawn, the attachment for the spinal manometer inserted and the spinal reading recorded without disturbing the remaining part of the apparatus. Five cubic centimeters of spinal fluid is withdrawn into the syringe, the stopcock turned to the right and the fluid expelled through the left outlet. The stopcock is then turned to the left and the syringe filled to the 5 cc. mark with air drawn through the sterile gauze. The stopcock is then turned to the center and the air injected into the spinal canal. The operation is then repeated until sufficient fluid has been withdrawn and subsequently replaced by air.

* Submitted for publication, March 20, 1930.

SPECIAL ARTICLES

PECULIARITIES OF BEHAVIOR

WANDERING MANIA, KLEPTOMANIA, DIPSOMANIA, PYROMANIA AND ALLIED IMPULSIVE ACTS: A REVIEW OF THE BOOK BY WILHELM STEKEL *

BENJAMIN KARPMAN, M.D.
WASHINGTON, D. C.

In the psychoanalytic world Stekel occupies a peculiar position. One knows well why others have dissented and wherein their stand differs from the orthodox; not so with Stekel. Adler left the camp because the concept of inferiority began to dominate his view on neuroses to the exclusion or subordination of any other; he was not willing to wait until Freud would provide in his system a place for his concept. When Freud was still searching for the ramifications of infantile sexuality, Jung was demanding with an ever-increasing insistence that due recognition be given to the higher, more cultural and more sublime aspects of the human personality; unwilling to wait for Freud to meet the demands, he left and developed a school of his own. And when Rank, outfreuding Freud, left, there were also numerous theoretical and practical differences. But in the instance of Stekel the differences were mainly personal; the differences of opinion that existed were minor and no greater than those usually found within any group.

It is whispered in Vienna that Stekel is the best freudian, and there is a good deal of truth in that. Like Freud, he works with the concepts of the unconscious, repression and infantile sexuality; like him, too, he believes in narcissism and the castration complex. If there is a difference, it is that Stekel, primarily a clinician, is not willing to accept all the theoretical superstructures that have been erected by the orthodox camp; he says that more clinical material is needed before one can go further in the matter of theory.

In this lies the difference in the attitude of the orthodox freudians toward the various dissenters. They smile condescendingly at Adler, dismiss Rank and express a certain respect for Jung; but toward none

* Submitted for publication, March 19, 1930.

* Stekel, Wilhelm: Peculiarities of Behavior: Wandering Mania, Kleptomania, Dipsomania, Pyromania and Allied Impulsive Acts. Authorized English translation by James S. Van Teslaar, New York, Boni & Liveright, 2 vols., pp. 633.

of these is there anything like a feeling of animosity. One need only mention Stekel's name, however, in the presence of an orthodox freudian, and there is sure to be an explosion and an effusion of bitterness and animosity which is clearly neurotic, for it is grossly out of proportion to the situation. Adler, Jung and Rank had an honest difference of opinion and left; God speed them! But Stekel has committed the cardinal sin—he has offended the Master; and so the faithful adherents, with their eyes on the master-Imago, transfer to Stekel the antipathy that Freud originally bestowed on him. He is laughed at, ridiculed, despised and belittled in every way possible and impossible; his merits are minimized as his faults are greatly exaggerated. Such an attitude is hardly calculated to bring about a fair-minded and honest appreciation of the situation. It is fully granted that his writings are pervaded with a spirit of conceit and vaunting adoration which often is annoying. All this, however, should properly be charged up to his personality make-up, and in itself need not detract from the value of the contributions as such. There are, to be sure, very few Spinozas among accomplished men. They are not known to suffer from pathologic modesty; most of them are megalomaniacs, implicitly or openly. It is granted further that Stekel is verbose in his writings; he seems to write more like a manic, and he often repeats. It is safe to say that each of his volumes could be abbreviated to one half, perhaps to even one quarter, of its original size with little loss and much gain. But even though he writes like an artist, as it were, there is none the less genuine scientific merit in his works. He is never dull or obscure, and in the analysis of a case he leads the reader step by step, so that his works are admittedly excellent guides to the study of psychoanalytic problems. Whereas in the majority of analytic writings one gets much theory and little case material, Stekel's conclusions are buttressed on a great deal of case material. His "Language of Dreams" is a classic of the first order, as is undoubtedly his "Anxiety States." He has made a host of fundamental discoveries (as the process of annulment, later rechristened as "denial," the neurotic mission, the significance of larval homosexuality, etc.) which are today incorporated in the theory and practice of psychoanalysis, but credit is admitted with great reluctance and obvious resistance. In spite, therefore, of many of his limitations, which are surely no greater than those of others, when the history of the psychoanalytic movement is written Stekel's name will loom large, for it is recognized that, almost single-handed, he has provided an abundance of most valuable clinical material, much of which is exceptional. He has done for psychoanalysis much more than Kraft-Ebing did for psychopathology. Just as Janet fills the gap between Kraepelin and Freud, so Stekel fills the gap between Janet and Freud. The significance of Stekel's contributions will be the

more appreciated when it is realized that psychoanalysis is still a more or less closed system, as yet un-understood and misunderstood by the medical profession; it will remain so until it is able to speak the language of general medicine. Where Freud emphasizes mass problems and seeks to uncover generic, even cosmic, principles, Stekel works in the main with individual problems.

In his monumental series, "Disturbances of Instincts and Emotions," he takes up serially a host of problems with which the practicing psychotherapist must deal every day. In the present volume, which is the sixth of the series, Stekel discusses a number of clinical problems spoken of as manias. These are impulsive acts centering around one specific aim. Since these manias or impulsive acts represent symptom-complexes rather than clinical entities and occur in combination rather than singly, the grouping of them together is for practical purposes only.

THE PROBLEM OF TICS

Of importance to the neurologist is the problem of tic, for tic originates in an impulsive reaction. The conception that tic is an organic disorder and a sign of degeneration has only recently given way to a more functional conception. From Trousseau to Charcot, from Meige and Feindel to Janet, this conception has gained rapid crystallization until it now finds universal acceptance. Janet believes that back of the tic there lurks an ever-affective mental image which he calls a "fixed idea."

The first extensive, and at the same time most authoritative, monograph on tic came from Meige and Feindel. They defined tic as a psychomotor disease presenting a coordinated purposive act, provoked in the first instance by some external cause or an idea, although later continuing to manifest itself without cause or purpose. Repetition leads to its becoming habitual and finally to its involuntary production at the same time that its form, intensity and frequency are exaggerated; thus it assumes the characters of a convulsive movement, inopportune and excessive; its execution is often preceded by an irresistible impulse and its suppression associated with malaise. The effect of distraction or of volitional effort is to diminish its activity; in sleep it disappears. The objective manifestation of tic is a clonic or tonic convulsive movement, the former presenting undue rapidity and increased frequency of the movements, while in the latter the duration of the contraction is prolonged. In spite of the disfiguration of the original movements, it is practically always possible to detect in them the original purpose. It is usual for several muscles to be concerned, and their anatomic nerve supply may be from separate sources. Like ordinary functional motor acts, tics are distinguished by a coordination of muscular contractions and repetitions;

they are preceded by a desire for their execution and succeeded by a feeling of satisfaction. In addition, the functional act is inapposite, sometimes even harmful; it may be described as a parasitic function. Tics are not accompanied by pain.

The authors further differentiated carefully tics from spasms, because of their external forms. Unlike tic, a spasm is a motor reaction resulting from a material pathologic irritation at any point in an afferent or efferent reflex, spinal or bulbospinal arc. The cortex is not involved in its production, unless it provokes these spinal and bulbar centers to activity. While a material pathologic basis appears to be lacking in tic, it is distinctly present in spasm. The movement of the spasm is extremely abrupt; purposive and coordinated features, confined to some nerve area anatomically limited, are lacking. Volition, attention, distraction or emotion fail to effect any modification of the phenomenon. No irresistible impulse precedes the manifestation, nor is it succeeded by a feeling of satisfaction. Sometimes the spasm is accompanied by severe pain. As a general rule, the patient's mental or psychic state does not present the anomalies met with so frequently among those who have tic. If the convulsive movement persists in sleep, it may be said with confidence to be a spasm.

The study of Meige and Feindel is mainly descriptive, and although they have reached some significant conclusions, they have overlooked many deeper psychologic determinants. They point out the lack of will on the part of persons with tic as an etiologically significant mental characteristic, as is also the infantile character of these patients. This, however, is only on the surface. Admitting the weakness of the will as an important contributory factor, insists Stekel, there is yet to be explained the reason for the weakness of the will as expressed in the neurosis.

In the course of time, the original gesture of the tic may and often does undergo transformation—"degeneration" of the tic (Janet). The authors further assumed a hypertrophy of the functional center of the brain, which may have been brought about by the weakness of the will (?). Janet, on the other hand, emphasized the psychic determinants and the psychic disposition, meaning thereby that the more or less rapid flow of mental images and thoughts is responsible for the tic.

The psychogenetic character of tic is proved by the fact that the trouble is curable through psychotherapeutic means, although tic is notoriously one of the most stubborn ailments and proves most refractory to every form of treatment. Brissaud employed mirror therapy and reeducation of the will, with varying results. Early hypnosis has been attempted, at times with satisfactory results. Raymond and Janet repeatedly attained success through the use of suggestion therapy. It must be admitted, however, that all these methods do not go into the

deeper psychic roots of the ailment. Therefore, the results are merely transitory and uncertain; these methods, unfortunately, fail in 95 per cent of the cases. Cured cases of tic are extremely rare.

Stekel goes further than the French workers, and believes that tic is symptomatic of a neurosis. Getting rid of the tic does not necessarily mean that the patient has been cured of the neurosis. The tic is the material expression of a mental conflict. It represents a thwarted impulse; it stands for an action arrested by the inhibitive images of the subject's primary personality; it is the expression of a wish, the mirror of a deed. The self is opposed by a counter-self, which betrays its aim through the tic. The aim is not expressed through a complete act, nor is the tic the caricature of an act, as claimed by Charcot, because it is almost impossible to interpret the original intent of the motion on account of the subsequent "degeneration" of the tic by reason of which the original sense of the intended action is gradually lost, leaving behind only vestiges which lend the tic its senseless and ridiculous character. The tic is really the rudiment of an action, giving the appearance of a caricature, but originally the motions of the tic were logical as well as meaningful. According to Jelgersma, the tic is a short circuit. The impulse avoids the path of conscience which represents the sum of all the inhibitions that stand between instinctive prompting and action; it breaks through either in the service of or against the conscience. The psyche appears to be excluded. The organic urge triumphs in a cryptic jargon which apparently has no connection with the consciousness.

Tics are extraordinarily common as masturbation substitutes. Thus one patient, addicted to masturbation, at one time indulged in this habit by rubbing a servant girl's handkerchief around his nose. Through the association of ideas between scratching the nose and sexual pleasure, one act became the symbol of the other, effecting the genitalization of a tic in the sense of Ferenczi. The association invested the reaction with strange value; it was also less dangerous and easier to carry out than masturbation. Other patients stick the hand in the trouser pocket and reach for the genitals with ticlike motions, without knowing it. A very widespread tic consists of scratching the anus, a habit which sometimes assumes serious proportions. An alleged pruritus ani serves as the justification for the habit of scratching the parts, but the evil persists as a tic even after the pruritus is entirely over. A whole series of repellent habits go beyond the limits of habitual mannerisms and, becoming automatic, assume the character of tic. Women who have given up the masturbation habit indulge in numerous substitutive mannerisms of a ticlike character.

The psychogenesis of a tic may be complicated; indeed, a single tic may be found to have many components, each having its own origin and

determinants, although one often finds that after the dissolution of the most important determinant the tic disappears.

The cause of tic, therefore, is not a weak will, as Meige and Feindel assumed; like all neuroses, the ailment is the result of a split will. The patient is dominated by the contrary, mutually conflicting wills. Along with the conscious (moral) integrated personality, another (amoral) is set up and, in spite of the former, breaks through, expressing its aim in the form of a tic. Many persons with tic themselves come to the same conclusion. As one said: "Two souls dwell in my breast: one with, the other without, tic." Another patient expressed motorially this split between good and evil in a wonderful symbolic form. The right hand carried on a relentless warfare against the left; sometimes the right grasped the left hand to avoid serious harm. Janet stated: "The tic patient, unlike the hysterical, has not lost the awareness of his compulsive gestures, but he has lost the sense of governing his motions." This dissociation is characteristic of tic as well as of all neuroses and many psychoses, especially schizophrenia. Hence the passing of tics into catatonia, noted by many observers.

The tic is thus viewed as an arrested impulse, which in most cases is the sudden outbreak of an instinct, restrained by moral inhibitions. The tic is likewise of lightning rapidity, impulsive, but its outbreak is systematized and symbolized. The automatic character of the tic creates the impression that the ailment is organic, which differentiates it from the compulsive mannerism to which it bears a certain resemblance. The compulsion neuroses are also due to an impulse, but in their case the impulse is not sudden, overwhelming and lightning-like in its manifestations. The compulsive act is preceded by a struggle between consciousness and the unconscious, between the integrated and the secondary self. The compulsive act is also linked with a death inference, such as is wholly lacking in tic ("if I do not carry out this or that particular action, some person dear to me is certain to die"). Of course, persons with compulsion neurosis may suffer from tics also. However troublesome and uncomfortable a tic may be, it seems to lack emotional accompaniments and its suppression is seldom and not so pronouncedly linked with anxiety, as in the case of compulsive and other neurotic acts. Suppression of the compulsive act rouses dread on account of the associated death inference, which is now always present in consciousness. Dreading the anxiety state, the patient with compulsion neurosis finally yields, unless he exhausts the compulsive in his thoughts (for instance, the impulse to strangle a woman), and protects himself against the compulsion thought through anxiety, confession or by formulation of a new compulsive neurotic overstructure.

In hysterical states one frequently sees ticlike motions, which must be strictly differentiated from genuine tic. These motions lack the lightning-like course, the twitching or the overwhelming spontaneity. They are still like the original motions, not yet transposed into the mnemonic picture of the tic, although they may become such unless they turn into some other form of transference and conversion.

It has long been noted that there is a relationship between tic and catatonia, and Meige and Feindel emphasized the passing of tic into catatonia. This is understandable when one realizes that the background is fundamentally the same. In tic there is a serious mental conflict which leads to a splitting of the personality; in schizophrenia, too, there is a similar splitting of the self into a number of partial selves. In tic, the partial self overwhelms a certain motorial path. In schizophrenia, the whole psyche of the conscious self is overwhelmed.

Related to this is the problem of the psychic background of stereotypies displayed in many cases of serious psychoses (paranoid, catatonic, etc.). Bleuler has long stated that the stereotypies are frequently symptomatic acts in Freud's sense. Stereotypies are closely allied to tics, but lack their lightning-like fugitive character. Nevertheless, in certain cases a line of distinction can hardly be drawn. Klasi has always been able to trace the meaning of these apparently senseless actions.

Stekel submits several interesting analyses of tic (there are very few reported in the literature) from which the following three case reports are abstracted:

Case 1.—A young attorney, married, suffered from the well known collar tic, frequently observed as a simple habit or stereotypic motion. Stiff collars gave him an uncomfortable feeling around the neck, and he constantly made the usual hand motion around the neck as if to test whether he had enough air to breathe. So long as this was done deliberately it amounted to a compulsion mannerism; but he repeated this motion automatically all day long without being aware of it. Besides, he suffered from an unusual eye tic. Both eyes turned synchronously to one side, the motion taking place with lightning-like rapidity. In addition, he suffered from a serious hysterical disorder of vision, not relieved by glasses, and this interfered a great deal with his individual and professional work.

He had been unable to wear stiff collars for a year, since his return from the army, and simultaneously the tic had appeared. Since his return, he had maintained sexual intercourse with his wife every night in spite of their daily quarrels and serious conflicts. He was extremely jealous. Often in his dreams he discovered his wife in breaches of marital faithfulness (wish fulfilment) and killed her. Such dreams amount to a pretext for latent hatred.

As a young man he was promiscuous, and was led into marriage through an unfortunate incident. It was the usual story: a love affair, illicit relations, pregnancy, a threat of court proceedings or marriage and hasty acceptance of the latter as the lesser of the two evils. He doubted (positively without reason) whether he was the true father of the expected offspring. The marriage was kept secret from his mother. Soon he went into the army. While in France he lived an abstinent life, in spite of his polygamous tendencies, because he clung to a super-

stitious belief that his wife would be faithful to him as long as he himself would remain faithful to her. On returning, he told his mother of the marriage (but not of the circumstances), and this evoked a vehement reaction on her part.

As a consequence, there arose within him criminal impulses directed against his family. He wanted to poison, or in some way to get rid of, his wife and the two children, but he was unconscious of this. As a warning and a defense there arose the tic—he was afraid of having to hang for the crime, and therefore constantly sought the assurance of having enough fresh air. The collar tic obviously stood in the service of the moral trend. His jealousy was a wish fulfilment and a projection of his own weakness. His eye trouble arose because he had resolved not to look at girls any more (really wanting to meet them). In spite of his resolution his eyes moved to one side even while he tried to gaze strictly ahead, thus bringing the tempting pictures within his visual sphere and seeing them with a part of his self. This tic stood in the service of the unmoral trend. The hysterical disorder of his vision was a conversion symptom; it expressed his inability to look at the truth.

The first dream he reported contained the solution of the conflict; it was a key dream.

He reported the following dream: "I am accused of having slain my mother. I know that I have not done so, because mother is still living. I face the court. Attorney N. (the same one who defended a woman slayer who was afterward hung) argues that it is not possible to have killed a person who is still living. Great controversy over that in the court room, it being alleged that mother has been slain, even though she lives. Enraged, I shout scornfully at the court: 'It makes no difference what you contend, I'll be sentenced anyway.'"

The problem of the dream was solved when it was known that he had stifled his love for his mother, causing her enough anguish to bring her nearer to her grave. "Mother" also stood for his wife, the mother of his children, whom in imagination he had repeatedly slain. The court room was his own conscience, against which he rebelled. Because of marriage he was no longer able to share his mother's life. His hatred was directed against his wife, the innocent cause of his "matricide."

Case 2.—Z. T., a Serbian, had suffered for two years from a tic of the left facial nerve. The tic was partly clonic, partly tonic. The right eye closed, and the right oral angle stretched to the right.

The family history was without significance beyond the fact that his mother and his uncle had some slight indications of tic. Physical examination revealed nothing abnormal, except slight mechanical hyperirritability of the left facial nerve.

He suffered from various anxiety states and compulsion symptoms. He walked about carefully for fear that this or that might kill him—a falling stone, an automobile, etc. In the house and in the street he was suspicious of strangers. He was careful when using a needle and thread for fear that the needle would injure his eye. He was afraid that he might lose his brother through an accident or an infection, and he worried about him if he came home late. Before going to bed, he fervently prayed for himself and all others, and before falling asleep he frequently had the sensation of falling into an abyss.

He was also subject to all sorts of compulsions. On the street he wanted to outwalk strangers, or he engaged in oracular mannerisms; he felt compelled to cross the street ahead of a certain automobile, otherwise some misfortune was in store for him. Everything must be symmetrical or even. He went through a complicated ceremonial while undressing. All the articles taken out of his pockets had to be set down in a certain order.

His sex life revealed moderate masturbation, including several episodes of mutual masturbation; he preserved good potentia and showed no predisposition to paraphilias. Until his return from the army, he had a new love affair every six months. As with many soldiers, homosexual components developed during the war at the expense of the heterosexual trends. Circumstances often compelled him to sleep in the same bed with his orderly to whom he was affectionately attached, but this never led to any overt acts. Now he despised women. What broke his faith was the brazen unfaithfulness of the wife of a certain friend, married but four weeks.

He slept soundly and he seldom dreamed. One stereotyped dream had recurred a number of times: He dreamed that his parents had died. All his dreams were concerned with accidents: He was injured in an accident, had a duel with his father, etc.

He had one sister and one brother. The sister was a chronic invalid. He had often wished her dead so that he might get her share of inheritance, and because he feared that after his father's death she would be a burden to him. The feeling attitude toward his brother was bipolar, an admixture of hatred and affection. During his youth he had hated him and had often harbored death wishes against him, partly because of the inheritance and partly because he was jealous of the preference which the mother clearly showed for the younger son. Now his attitude had changed into love. They slept together, and he was much concerned over his brother's welfare.

The roots of his anxiety were traced to the war. He was a Serbian, who had served in the Austrian army, and who had been confronted with a painful dilemma. He had faced the Italians and among them probably also Serbs, his brothers. For this reason he had always discharged his arms into the air. Once he found himself in the situation of having to shoot an Italian. It was a horrible experience for him. He killed him, and he felt as if he had slain a brother. As he recited this, he jumped up horrified and showed how he had shot down the Italian. This incident disclosed that the tic reproduced his facial mimicry during his act of shooting. He shut one eye the better to aim with the other, while his mouth was distorted to one side. His tic was the fixation of a shooting fantasy; it dramatized the intention of shooting down his brother. The criminal impulse expressed itself in the tic; the various anxieties for himself expressed his fear of retribution, while those for his brother were overcompensations. The homosexual fantasy was disclosed by the fear of sticking a needle in his eye (anus, the "brown eye"—Freud's transposition from below).

Case 3.—T. K., 32 years of age, an engraver, suffered from a peculiar mouth tic. When he was about to begin to talk, and sometimes while talking, the lips protruded forward trunklike, his face contracted to the left, and saliva gathered in his mouth. Then, unable to continue, he lowered his eyes in confusion.

The various components of the tic seemed to have a different and an independent origin. The first symptom of the trouble developed at the age of 16 and had reference to a teacher who was antagonistic toward him, and because of whom he became shy and retired and developed confusion and lowering of the eyes. A little later, the difficulty began to center around the mouth; it consisted of a feeling of tenderness and weight in the lips. Gradually this began to interfere with swallowing, and became complicated by an excessive flow of saliva and a feeling of strangulation around the throat. Finally, the face began to deviate to one side, and at last the tic appeared spontaneously and no longer in connection with any attempt at speaking.

Even a cursory review of his life revealed a number of significant homosexual trends. Physical development was tardy, and the first pudendal hair did not appear until the age of 17 (the age when the trouble began). He was the oldest of six brothers. He had always wanted a sister, but had none. Girls he had regarded as dirty, inferior creatures; later he regarded them with indifference. At 17, he met a man at a bathing establishment who taught him to masturbate and also urged him to undertake a homosexual relationship. He rejected the suggestion, but since that time had been aware of a longing for men. Subsequently he was interchangeably conscious and unaware of his homosexuality. He had had a number of opportunities but he had not yielded to the wishes. During the period from his twentieth to his twenty-fourth year, he occasionally resorted to prostitutes. At 24, he acquired gonorrheal infection which caused him deep humiliation and drove him to attempt suicide, which narrowly failed of its aim. A more conscious recognition of the homosexual trends operating in him brought about a marked improvement, so that he lost the discomfort and was able to look more steadily at persons while addressing them.

Next he recalled as a childhood memory that between the ages of 6 and 10 years he had often endeavored to reach his penis with his mouth. This was found to have a connection with the mouth trouble. Until his tenth year, every time a baby arrived in the family he sucked at his mother's breast along with it; he was passionately fond of doing this. Frequently, the mother, in a spirit of pleasantry, sprayed her breast milk into his face. His mother also used to kiss his penis as a means of pacifying him. In his dreams he often saw a milk-maid or a milk-wagon along with his brothers. Milk, spermatic fluid and saliva represented the same concept in his dreams. This led to an understanding of the flow of saliva in his mouth. The motions of the mouth expressed the wish of carrying out fellatio on the person whom he addressed or of sucking at the person's breast. The gathering of saliva represented the symbolic gratification of his wish by suggesting the oral perception of the flow of milk and of the spermatic fluid ("the eternal suckling").

The foregoing analysis brought about an improvement, so that the spasmodic tic motions became limited to the anterior parts of the mouth, and their compulsive character was markedly allayed. Further associations disclosed the relationship between the muscular spasm around the mouth and throat and his jealousy and hatred of his brothers. In his dreams, his father persistently discriminated against him and in favor of his brothers. This, in the dream, aroused his anger and produced an unpleasant sensation in the mouth and throat. The spasmodic rigidity of the lips and the sensation of tightness in the throat represented the oaths and death wishes which he would like to hurl against his father and against the world at large because it disdained his pleasure craving.

Next and last were revealed some peculiar habits during earliest childhood (from 4 to 7 years). He was particularly interested in defecation, less so in urination. During defecation he tried to enhance his pleasure by tickling the anus with his fingers. He also pressed the penis in a position to reach the anus and often tried to reach the penis with his mouth. He watched with pleasure his playmates in these acts, and he tried to attract their attention while defecating. This became a regular compulsion and led to the habit of imitating with his mouth the act of defecating. First he stretched the lips forward, to imitate the protrusion of the mucous membrane, and then he forced the saliva through to represent defecation. With the behavior of his mouth he demonstrated to the whole world that the anus was his most important bodily region; with his tic, he rehearsed the act of defecation so that the man who observed him would fall in love with him.

The final point was the distortion of the face to one side during the tic, his last symptom. During his attempt at suicide he pointed the revolver to his right temple and also distorted his face to the right. Further associations disclosed that he harbored mother-body and father-body fantasies, and that his attempt at suicide was but an expression of intra-uterine regression.

The mechanism of the tic was thus unraveled, but the driving motive, the homosexuality, was not thereby annulled. As long as this was actively present, a relapse was to be feared. The vehement fight against homosexuality brought forth as one root the infantile feeling-attitude toward his father, specifically the wish of serving the father as a wife substitute. He dreamed of his father shooting at him, the bullets tickling him; of his father assaulting him per anum. He entertained the same wishes against his two older brothers (fellatio and pederatio). But identification with his mother proved to be the most important root, and in a double sense. On the one hand, he wanted to be the wife to the father, as his mother was; on the other hand, he was in love with his mother and jealous of his father. His dreams often dramatized his feeling-attitude toward his mother. He often wished to assault his mother while she was asleep or dead. The uncovering of the necrophiliac root brought about a marked improvement in the patient's condition. The final effort was that he not only rid himself of the tic, but was also about to make a better heterosexual adjustment. The tic was a self-betrayal; what he denied himself in actual life, he staged with his mouth.

Looking over the cases cited, one finds that in every one of them it was possible to ferret out the unconscious motives and, by bringing them into the conscious, to relieve the tic. The language of the muscles became superfluous after it was replaced by frank thoughts. The strange energy which compelled the patient to carry out certain motions was no longer foreign. By entering consciousness it became a governable part of the personality. The repression was replaced by condemnation (Freud).

DROMOMANIA

The impulse to wander or dromomania may be of two types: the centrifugal form or "fugues" (temporary absence), flight from home, and the centripetal form or flight back home (nostalgia). Back of these flights are difficult and unadjusted emotional situations. Preceding the attacks is a torturing restlessness which the patients themselves are unable to explain. The instance is recorded of a girl who would run away from home. She had strong leanings toward the father, and she fled from the house because she could not stand her mother's way of treating her father. In centripetal dromomania, the longing for home is accompanied by feelings of anxiety, which may become so strong as to interfere with clear reasoning.

SOMNAMBULISM

Sleep walking or somnambulism is a phenomenon that may have a complicated etiology behind it. As Stekel puts it, sleep walkers are dreamers with their eyes open, struggling with temptation repellent to

consciousness. Unfulfilled wishes, which cannot be realized during the waking period because of repression, are sometimes, when the impulse is overwhelming, allowed full expression during sleep when the conscious inhibitions are removed. Thus crimes or deeds symptomatic of them may actually be committed during sleep and dreams, and the motivating emotion, be it jealousy, rivalry, ambition, hatred or some other, may be given free outlet. Similarly, sexual impulses may be given expression during sleep, and "nocturnal doings" (intercourse between brother and sister, parents playing with the genitals of their children, etc.) covered by complete amnesia.

DRUG ADDICTION

In drug addiction Stekel sees an expression of a neurosis in which the person attempts to overcome a pathologic depression or to still an unbearable anxiety through the use of a chemical crutch. It is this pathologic emotional state that, to begin with, drives the person to the use of drugs, and it is mainly because of this that the withdrawal of the drug brings on a severe anxiety—for in the absence of the drug the original depression and anxiety return. The so-called drug cures are seldom efficacious, for they do not touch the fundamentally psychic aspect of the problem. A deeper probing into the mental life of drug addicts reveals that they have severe neurosis with all sorts of distorted or exaggerated emotional attitudes, abnormal sex life, sadomasochistic reactions and a luxuriant fantasy life that abounds in all sorts of criminal fancies, which, however, never come to fruition but are followed by a torturing sense of guilt, perhaps the fundamental cause of anxiety. Although much more difficult of approach than ordinary anxiety neuroses, drug addiction is distinctly approachable through psychotherapy.

Case 4.—The patient was given to wandering, changing places or drinking. He grew particularly restless during the month of April. Analysis revealed fixation on a sister, with childhood episodes of coitus with her on several occasions. Her marriage provoked in him a tremendous jealousy reaction at her husband, and precipitated him into an acute panic; on the eve of the ceremony, he drank himself senseless. He then fell in love with a girl (transference), but after attempts at coitus found himself impotent (thoughts about the sister). The impotence was followed by depression. In order to find out whether he was really impotent, he tried masturbation and was amazed at the strength of the erections (fantasies about the sister). Obviously the depression was not due to impotence, but to moping over the loss of his sister. April became the month in which he celebrated the wedding by getting drunk, the period being dedicated, so to speak, to the memory of his old relationship (the neurotic's secret calendar).

Case 5.—A man suffered from an anxiety neurosis, with typical attacks of heart symptoms and fear of death. Recourse was taken to carbromal before bedtime lest the spells come on him during the night; without it, the fear of the spell would keep him from falling asleep. The personal history revealed an

apparently normal sex life; he had sexual intercourse with his wife daily, a circumstance in itself suspicious, since the sex life of a normal person is more or less periodic. In neurotic persons, however, it is not infrequently observed that regular daily release of sex is indulged in on account of the hidden fear of some neurosis; so that the question in each case is less the frequency and extent of sex gratification than whether the person has found the form of gratification which alone is adequate for him. Further study in this particular case revealed a tremendous jealousy toward the wife, fixation on the family and a certain predisposition toward homosexuality. Behind this were found marked voyeur tendencies. In particular, he was passionately fond of watching children and women in the act of defecating; for this reason, he frequented urinals and looked for convenient holes through which to peep. For fear that this would eventually bring him into conflict with the police, and to suppress his paraphilia, he married. The desire, apparently no longer present, still asserted itself through these attacks; in order not to yield and not to dream the patient took the drug.

In a case of barbital addiction, in a man, aged 40, an intense affection for his daughter was brought to the surface. The narcotomania set in after a period of extreme sleeplessness.

GAMBLING

Gambling that passes the limits of normality is regarded by Stekel as expressive of a neurosis. A man becomes a victim of gambling when normal interests are lacking. Back of every game is a conflict; it is a fight for the supremacy of one's personality. However meager his talents may be, inwardly every man considers himself as, or at least would like to assure himself of, being capable in some realms; in such cases gambling appears distinctly as a compensation for feelings of inferiority. During the game, players undergo remarkable transformation. Because of a consciousness of mastery, a quiet, unobtrusive man changes into a courageous, self-conscious person who exaggerates his successes and minimizes those of others; the kindhearted man turns into a sadist, teasing his antagonist; the taciturn turns loquacious, the game, like alcohol, unloosing his tongue; spendthrifts become greedy and tremble over the loss of trivial sums of money to which under other circumstances they would pay no attention. Many so-called respectable persons mix cards in a tricky manner and often deliberately falsify accounts and reckoning without troubling their minds about it.

Case 6.—E. G., aged 45, gave a history of an unhappy marriage. His wife appeared to be frigid, and to satisfy his craving he finally effected an arrangement with her whereby he would give her money every time she would allow him to have sexual intercourse with her—a remarkable form of marital relations. Suddenly he became jealous of her, began to have strange dreams in which he saw his wife having intercourse with other men. He began to watch her closely, with the result that he discovered that she had five lovers, one of whom she showered with gifts which she bought with the "pin money" he had to pay her for intercourse. A terrible quarrel followed; at first she made a denial, and later a plea for forgiveness and a promise never again to break her marital vows. The affair shook up the patient terribly; he began to doubt whether their two children were his, although she would reassure him a thousand times; he would gaze at

them for hours, scrutinizing their features. He now began to suspect—perhaps not without reason—that his wife had had a lover before marriage; hence, perhaps, her tremendous resistance during the bridal night, by which she meant to arouse his desire more keenly and deceive him about the loss of virginity.

The history disclosed that at the age of 24 he was engaged to a girl who confessed to him one day that she had betrayed him during his absence of two months. Two years later, he fell in love with another girl who too, after a while, confessed to him that during his absence she had yielded to another man. He began to imagine that he was unattractive and could not hold a woman's interest. Therefore he had to pay his wife; he could get only prostitutes by paying them for service rendered, but could not conquer a woman. When the disappointment with his wife came, it broke his pride altogether. But he grew jealous over his wife's past; she had to confess everything to him. "I suffered nameless tortures, but at the same time, it roused me sexually. I enjoyed while I suffered; I suffered while I enjoyed. The saddest part was that during sexual intercourse with her my thoughts would wander off to the other men. It would occur to me that they were stronger and perhaps knew better how to love—and my erection was hopelessly over. I was not completely impotent with my wife. I demanded faithfulness from my wife, but I could give her no longer that which she hungrily wanted and expected of me alone—for now she wanted only love and to purge herself clean. I could not do it. . . .

"And then I began to drink and gamble. I sought forgetfulness and peace, surcease from my troubled thoughts; whereas formerly I had never been to a race, I began to drift more and more into the gambling habit, completely neglecting all my other work. Besides the races, I play cards and other games of chance. I had lost my interest in my wife or any other woman. But for an occasional pollution, I would think I had no sexual life. At the races, when the stakes are high and my situation is endangered by a loss, my anxiety is distinctly adumbrated with pleasure. At school, I felt likewise about my lessons; and some school tasks, particularly dreaded, ended with pollution. During the races I have almost had the same experiences. . . . I have also developed a fever of buying, buying things right and left for the sake of buying—it seemed as if I meant to throw away the easy money. . . ."

It was plain that when the patient could not hold women, there was something about his behavior or sexuality which repelled them. It may be surmised that, by reason of some fixations, he instigated his defeats. The analysis, which turned out to be enormously difficult, brought to the surface some remarkable features. He had masturbated since childhood, utilizing his mother's and sister's underthings, taking these with him to bed and from time to time also putting them on (fetishism). Afterward, he stole pieces of underwear to take along on journeys. The odor roused him so tremendously that smelling the clothes was enough to induce an orgasm; the odor of worn drawers was also the best soporific to induce sleep. The conjugal relations between his father and mother were not of the best, the mother having deceived the father regularly with a number of lovers. The boy knew it and bore a grudge against his mother; it took him a long time to overcome his mother's prostitution complex. Returning home one forenoon, he found his mother's bed in disorder with suspicious looking spots. Nobody was home. He put on his mother's nightgown and fell into an ecstasy such as he had never experienced in his life (fantasy of relations with the mother and taking the place of her lovers). He swore never to masturbate again (the incest barrier) and kept his oath. He burned up the old underwear, thinking to rid himself of the paraphilia. Only the parallelism, mother-prostitute, made it possible for him to

treat his wife as a prostitute and pay her. So long as the identification was maintained in fantasy and merely by the act of paying her, he remained potent. But the discovery of the actual facts lent an extraordinary reality to the equation: His wife turned out to be a real prostitute. It brought the incest idea too close, and in defense he became impotent.

The question arises whether, to begin with, he did not choose his sweetheart among frivolous girls. This surmisal is corroborated by the analysis. All three girls with whom he had fallen in love were of the flirtatious type and did not enjoy a good reputation. That precisely was what had drawn him to them (emphasis on the prostitute part of the equation), although at that time he exacted strictest faithfulness from his love objectives (emphasis on the mother part of the equation; hypothetically he was exacting the same condition from his mother). From the moment his wife became completely identified with his mother, his potentia was gone. He regressed to his infantile ideal. He began once more to visit his sister, whom he had badly neglected after his marriage, and he devoted himself to her. The impulse to masturbate arose again (incest fantasies), but an irrepressible feeling of shame prevented him from indulging. On the other hand, he came near stealing pieces of underwear from his sister.

His wife's unfaithfulness may be accounted for, in part at least, by the fact that the patient was not very potent; he was inclined to ejaculatio praecox. He always carried on sexual intercourse in the dark; his wife, who was a pronounced exhibitionist, he never saw naked. He always addressed her as "my sweet little mother." He held on to her because she was the mother of his children (identifying himself with his children, and his wife with his mother). During the period of her pregnancy he was very potent, obviously because various infantile wishes were thus fulfilled.

On reviewing the lessons taught by this case, one notes that the first and apparently the most important motive, the love disappointment, was merely a superficial determinant. If every one who was disappointed in love turned into a gambler, there would be many more gamblers in the world. In this patient, it brought on a regression to infantile feeling-attitude and awoke particularly his lust for his sister. The analysis further disclosed that every big gambling venture became an augury: (junctim) "If I win the game, it means that I gain my sister." Often the degree of excitation did not correspond to the amount at stake; it was a transference of affect. The real prize at stake was his cryptic erotic aim; something very precious. As a proof of this assumption looms up the fact that he bet on a certain horse against all reason, merely because it bore his sister's name; he was convinced that this horse must bring him luck. The subsequent course of events corroborated this morbid love of the sister. As he became wealthy he built a large villa for himself and, against his wife's will, took his sister and her husband to live with him.

KLEPTOMANIA

Stekel looks on kleptomania as a symptomatic act which is only one of the many links in the large chain of the neurosis present. The motive back of it is unconscious and is expressive of some unsatisfied emotional situation. It is owing to the symbolic nature of the act that the relief, because only substitutive, is only temporary, while satisfaction is derived from the commission of the act itself and not because of the economic value of the article; for not only is the article stolen often of little or no value to the kleptomaniac, but quite universally it is thrown

away immediately on the commission of the act. Shoplifting belongs in this group. The irrational nature of the act comes more into evidence as one notes that it is often committed by women who are well-to-do, when the economic motives are obviously lacking and the risk incurred is out of proportion to any gain to be made. It is also significant that during the act the patient is in a sort of an emotional daze, as if he or she were in a different world. Stekel's thesis, which he supports by a large number of carefully studied cases, is that ungratified sexuality looms large as an etiologic factor in kleptomania and allied reactions, and that on the release of this, the kleptomaniac act, of which it is but a symbol, disappears. Naturally, one finds all sorts of psychosexual difficulties here, as in many other neuroses.

Kleptomania may be associatively related to frustrated love, jealousy and rivalry; to impotence, frigidity and forced abstinence; and in deeper cases, to complicated paraphiliac situations, as obligatory auto-erotism, homosexuality, sadomasochism, fetishism and other fixed ideals. Occasionally, one finds cases of kleptomania in which the commission of the act is followed by an orgasm. But even if this is directly or obviously absent, the presence of secondary emotions—anxiousness, excitement, somatic tension and its release—is not at all unlike, and suggests the presence of, a sexual situation. In many women the reaction appears or becomes exaggerated during menstruation or pregnancy.

Case 7.—A married woman, aged 37, gave a history of many hysterical reactions, attacks of anxiety and serious suicidal attempts. Analysis revealed that she was hypersexual, much dissatisfied and suffering because of her husband's apathy and occasional impotence. She had dreams about her husband being injured, dying, etc., and the dreams were accompanied by considerable anxiety. Once she discovered him having relations with another woman, which stirred her up. Her husband's younger brother, who was married to an artistic wife who reproachfully boasted to the patient that she married for love, had begun to pursue the patient with his attentions. The patient then began to steal from her husband and to decorate her house with artistic objects—symbolically trying to get ahead of her sister-in-law, of whose husband she was jealous. The unconscious wish to be rid of her husband, expressed in the dreams, fits in precisely with the tendency, expressed by the thefts, of putting herself in the rôle of her artistic sister-in-law who married for love. Here, too, the misdeeds show themselves to be, above all, symbolic acts, although the symbolic interpretation alone is not enough.

Case 8.—A girl, aged 22, since childhood had been stealing from friends and stores; in her own words, "her impulse was stronger than her will power;" but her conscience did not trouble her over the taking of things. She would use the stolen money mainly for gifts for others, to buy their good will, because her queer moods and deeds repelled them.

Of her sex life, the significant points are that she never felt any attraction to men, although craving their attention. At about the age of puberty, she became attached to a woman teacher, nine years her senior, in a friendship which later proved to be a sensual relationship; on her part, however, the patient was rather passive and unwilling and only submitted as a condition of friendship. She claimed

lack of affection for her mother, but there was much more behind it. "Mother's love was divided between a friend of hers and myself. I, an egoist, wanted it all, and that's what the teacher gave me: care, patience, forbearance of my faults. Back of her severity I perceived love. She was a mother to me, while my own mother punished me when I tried to get nearer to her. She would repulse me."

It is obvious here that the patient's impulse to steal was due merely to an ungratified craving for affection and not to repressed sexuality. She loved her mother and was jealous of the latter's friend; failing to enjoy the mother's love, she undertook to avenge herself at the mother's expense. This also drove her into homosexuality; connections with homosexuality, especially attachment to mother or sister, are frequently observed in female kleptomaniacs. This patient became a criminal because she hated some one. (Whoever hates another, hates the whole world.) As is usual in such cases, stealing and giving away go together because it is the act of stealing that satisfies the particular emotional need and not the object proper for which there is no need. Further, the act of giving may serve a compensatory function by enhancing the ego feeling previously lowered by the theft. And it may also have an erotic value: The subject wants to give himself away and substitutes some object in transference. Many crimes, including murder, are carried out by persons who have been badly treated by their parents and who thus give vent to their feelings on some one else.

PYROMANIA

Except for the difference in the act proper, pyromania has about the same basic motivation and clinical significance as kleptomania. Like the latter, it may be a part of a larger neurotic constellation, with a multiplicity of motives behind the act. A prominent etiologic factor in pyromania is the motive of revenge, for arson is an act of hatred; but as in other neurotic reactions there appears to be a gross lack of correspondence between the provocation for the hatred and the deed itself, for the alleged revenge motives appear petty and trivial. The particular cause for revenge varies; it may be due to being hurt, being unhappy in love or to humiliation, revenge against the faithless lover, the purpose of revenge being to inflict shame on the family or on the individual. The impression that one gains from the study of persons with pyromania is that at the time of committing the offense they are unhappy and dissatisfied, tortured by morbid fears and living in a state of remarkable tension, and that the arson is an explosion meant to put an end to an unbearable situation. Often the arson is committed either previous to or during the time of menstruation or during pregnancy, which suggests an endocrine basis.

Further, according to Stekel, pyromania belongs to a particular developmental phase in psychosexual development. The awakening and ungratified sexuality often impels a neurotic person to seek a symbolic solution of his conflict between instinct and reality. Many persons with pyromania have enuresis, and there is a definite relationship between masturbation and pyromania. The fire may stimulate one to masturbate or may be intended to free one from the habit (arson is often carried

out during a period of abstinence), or masturbation is resorted to as a defense against fire. Fire becomes a symbol of sexuality. Paraphiliac persons, prevented because of infantile fixations from finding the form of sexual gratification that alone is adequate for their needs, are always ungratified, and their impulses must undergo a transformation of objective before finding an outlet. There is a relation between impotence and frigidity, on the one hand, and pyromania, on the other, because the former conditions, like masturbation, are traceable to an unrealized or unrealizable love objective; lack of potency in one field apparently is compensated by an expression of potency in another. Homosexual, sadistic and other regressive motives may be at work. Orgasm may be achieved during a conflagration.

Case 9.—A married woman, aged 31, complained of the compulsive thought of some day setting her bed or home on fire; in addition, she also suffered from a fear of harming herself or committing suicide. Once she set fire to her brother's bed. The attempt was harmless; nevertheless, she had the hallucination that the bed was already enveloped in flames. She carried marked ideas of revenge against her sister whose home she wanted to set on fire.

In her daily behavior she displayed a number of parapathic symptoms. She lived in a trance-like state and utterly lacked will power; unless others decided for her and told her what to do, she did not accomplish anything. Her father had been extremely severe with her, and without his permission she could not take a step. Now her husband took her father's place, the patient thereby avoiding personal responsibility as regards either domestic affairs or the care of her children. She even complained that she dreaded the children because while playing with them she became sexually excited; she struggled against the impulse of masturbating the boy and of sticking something into the girl's vagina.

As a child she was much preoccupied with fire. Fire and scintillating ornaments excited her sexually. Since the age of 4 years she had played with fire, starting small fires, but usually putting them out.

Her sexual awakening was precocious. She remembered experiencing pleasurable feelings when her father rocked her on his knees (touching at once two erogenous zones); she had since longed for this experience during which she felt herself defenseless. She began to indulge in auto-erotic practice in her sixth year. With some interruption, she had continued the practice until recently, when she gave it up; her present abstinence was in part responsible for her symptoms. With her brother, two years her senior, she had indulged in mutual masturbation and even attempted coitus; she still preserved her affection for him. Her homosexual components were strongly developed. She had always been fond of her sister, five years her senior, and when they slept together, as often happened, she would experience great pleasure. At 15 years of age, she became very pious and thought of becoming a nun, but this lasted only half a year. She also showed strong narcissistic tendencies. She was genuinely in love with herself and frequently stood in front of the looking glass, masturbating; she was always eager to hear compliments. At 16 years of age she was seduced by an army officer, who deflorated her on his lap; in this position of sedens supra sedens she became so excited that she lost control of herself and yielded. (This recalls the experiences that she had with her father.) The experience with the officer marked the onset of a number of parapathic symptoms. She was afraid of pregnancy and

of infection and resolved thereafter to be virtuous; this oath she kept in spite of numerous temptations, not the least of which was the example set by her sister, who was a frivolous woman and frequently betrayed her husband.

She married at 18, mainly because of the insistence of the family, much against her own will. On the bridal night she was completely anesthetic and had since remained so; she went through the ordeal of sexual intercourse as a matter of duty. Frequently she wept because of homesickness. Then children came, but she had no maternal feeling for them. In her daily attitude she had remained a child; she clung to all her childhood underclothes and souvenirs as precious relics, and played with children and even with dolls; she also said the child's prayer at bedtime.

She turned pious again and compelled her husband, who was a Jew, to undergo baptism. She looked at his membrum and, because it was circumcised, thought it was horrible. Although admired and courted by many men, she remained faithful to her husband, and always reported to him the various courtships. Once she nearly yielded to a man who assiduously tried to win her intimate favor. As her horse had run away, she was obliged to ride with him on his horse. She sat in front, practically on top of him, in a situation critical to her (recall the early experiences with the father). Then she jumped off. He kissed her passionately, and membrum suum dedit in manum. She had already attained her highest sexual acme and that saved her. She ran off like a wild creature and began to shriek from sheer delight. Suddenly she recovered from her acme. The astonished man let her go. But she continued to think of his membrum which she stated was much bigger than her husband's.

It was on account of these fantasies, which made her dwell in nebular regions, that she was absent minded. She carried on a genuine dream life during her waking state, and also slept many hours. Sleeplessness and sleepiness alternated with her. Some time after her marriage, her brother came to live with her. His presence fostered new fantasies. She craved a repetition of her infantile episodes with him. Her absent mindedness grew worse. Feeling herself entirely a child, she annulled her marriage, and with it annulled everything else she had gone through. In that state of mind she set fire to her brother's bed.

After the analysis, the morbid symptoms disappeared. Her love for her husband was awakened. She suggested to him to carry out congressus sexualis in positione supra sedens (reminiscent of the pleasurable experiences with her father). In this posture she began to experience sexual acme, and the evil path was thus broken. Her pyromaniac impulses also disappeared.

CRITICAL COMMENTS

Stekel's theoretical contributions are neither large nor very significant. Essentially a clinician, he dislikes theoretical excursions. In the opening chapter he discusses the problem of instinct, dividing it in the orthodox manner into two main categories, the self and the race preservative instincts, respectively. He further states that in accordance with his law of bipolarity (not explained here), which requires that every instinct has its positive and negative aspects, the race preservative instinct is the life instinct since it guards over the future of the race, while the self-preservative instinct is the death instinct since it is concerned with the current activities of life but ultimately leads to death. Stekel does

not submit any proof of the correctness of the concept, which is expected to be accepted as a dogma or an axiom. Aside from questioning the usefulness of pigeonholing the concept of instincts into two types, the reader may well question further the thesis of bipolarity as being so universal, and in particular whether, to satisfy the law of bipolarity, he is under obligation to accept the death instinct as opposed to the life instinct. It is known that for some time Freud has been struggling with this concept, but in spite of many brilliant arguments has not yet given a satisfactory statement of the problem. Is one to understand by the death instinct the unconscious striving toward dissolution, and that it is present in every human being alongside the urge to live? This would seem to violate the lesson of biology that the function of life is to continue itself, and that individual death is merely one of the links in the great chain of life. It may be the law of nature that cats should eat birds, birds should eat worms, etc., but who would believe that some unconscious instinct of death drives the bird into the mouth of the cat? One can readily concede that life may sometimes become so unbearable that death is a great relief, without taking recourse to the idea of a death instinct.

Even in matters of theory, however, Stekel can be sharp and pungent, especially when the argument seems to contradict his clinical experiences. In discussing the psychopathology of pyromania he takes issue with the views of Schmidt, a pupil of Jung. Schmidt was dissatisfied with Stekel's causal explanation of the emotional and paraphiliac barrier and preferred Jung's "final" explanation. He therefore proposed the hypothesis that conceives arson as a regressive symbolic act, which attempts to dissolve the ever present libido stagnation by an archaic and therefore worthless effort at sublimation. Basing his consideration on Jung's energetic conception of the libido, he stated that the libido energy is dammed up and then suddenly released, and is thus responsible for arson. In further support of this theory, he stated: "Just as the primitive folk compared fire friction with sexual congress, the libido release κατ ἐξωχῆν, so does our unconscious use the fire as the symbol of the gratification-craving, release-scorching libido."

Stekel, endeavoring to reduce the most complicated problems to the simplest terms, objects to such formulations as that of Schmidt. He states that he has steadily avoided Freud's libido theory, together with its energetic formulation and its "archaic delineation" by Jung, as in his opinion it is not required at all. "We know," he says, "that there is sexual instinct, we know that it manifests itself as desire (libido), but we cannot weigh this libido, nor fathom it with a yard stick; it remains an obscure, hypothetic concept. Jung's libido stagnation is nothing more than ungratified sexuality, which, perhaps, also involves a chemical component (the accumulation of sexual substances). But persons may

get rid of these sexual substances by regular intercourse or through masturbation and still remain ungratified. These are the individuals who are neurotic because they have not found their adequate, to them alone peculiar, form of sexual gratification. This constellation leads them into a conflict between craving and inhibition." One may express the situation in energetic terms, as Gross and Stekel did before Jung. Otto Gross expressed the energetic concept very forcefully: "The affective (emotional) energy is transferred from the unconscious sexual motive to the crime motive. After this transference becomes fixed, the crime motive may become the symbol for every wish for sexual gratification and take over the whole impulsive energy of sexuality. And it is this shifted affect then that determines the morbid compulsion."

Schmidt emphasized that the assumption of a sexual wish is not enough to explain the tendency to pyromania and, to support his statement, submitted cases in which the incendiaries, shortly before seizing on the notion of setting a fire, had indulged in sexual intercourse; he concluded, therefore, that "there could hardly be a question of repressed sexual wishes." Such statements on the part of Schmidt reveal, according to Stekel, not only a narrow conception of sex, but even ignorance of clinical knowledge. There are men who have pollution after repeated intercourse and men who must masturbate after repeated intercourse, just as there are women who remain ungratified in spite of repeated indulgence in sexual intercourse. These are disorders of the sexual acme in men and women. For it is not merely a question of intercourse; it is a question of sexual acme, of the adequate form of sexual gratificaiton. When arson is committed in spite of successful (?) sexual intercourse, there should not be, if Jung's contention that sexual intercourse is a "libido releaser κατ ἐξωχῆν" is correct, any damned-up libido and therefore any occasion for arson. The energetic conception as thus advanced is reduced ad absurdum and is indefensible.

Although the essential purpose of Stekel's contributions is the elucidation of the psychic background underlying neurotic reactions, he is not unmindful of their social significance. Originally erected as barriers against antisocial behavior, these neuroses appear to be expressions of broken compensations; the socialized behavior is changed into antisocial conduct because the individual is unable to solve his emotional problems in favor of the family or the social group in which he lives. This gives one a hint as to the possible psychogenic source of antisocial reactions in general and criminality in particular. Even though Stekel believes that there is a vast difference between the motivating emotions of the person with kleptomania and the common thief, in reality it is often difficult to draw a line; a deeper study of the common thief reveals him, too, as having been originally conditioned on emotional situations which furnish the driving power for the criminal act. At any

rate, the work furnishes a suggestive stimulus for further research along this line. Perhaps it may yet be discovered that criminality is a form of neurosis and that the treatment of the criminal may be approached through psychotherapy.

Toward the forensic aspect of the problem, Stekel maintains the view that since kleptomania and pyromania are unconsciously motivated modes of behavior, the persons guilty of them cannot be looked on as criminals, but as mentally ill people who must be treated and not punished. That many such persons come from socially poorly adjusted families and from homes that are anything but harmonious, suggests to Stekel that the remedy lies not only in psychotherapy but in a radical change in the educational system, and perhaps even in a better social economic system. He believes that the emotional cost of the World War was perhaps even greater than its economic cost. In its wake epidemics of pyromania broke out and criminality in general became rampant, for the war loosened unchecked the crudest and most primitive instincts.

As thus envisaged by Stekel, neuroses have their individual psychogenic settings as well as their social economic background. Such a view is a wholesome relief from the narrow paths in which the present psychoanalysis is moving. The understanding of human behavior cannot be monopolized by a one-sided study. Neuroses must be studied as expressions of the total personality and particularly with reference to the social medium in which the individual functions. Only such a study can bring about a fruitful combination of their treatment, cure and prevention.

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PROGRESS IN PSYCHIATRY

V. EIDETIC PHENOMENA AND PSYCHOPATHOLOGY *

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In the development of psychiatry it has rarely happened that the description of an isolated phenomenon of experimental psychology has had a great influence on psychopathologic thought. It is therefore of interest to see that from the work on eidetic images which has been done in recent years, many inferences have been drawn with regard to psychiatric problems. Extensive claims have been made as to the light which these studies of the experimental psychology of visual images can throw on questions of pathology. It seems worth while, therefore, to survey briefly the field of these studies and to examine to what extent they actually are advancing the solution of psychopathologic problems.

It has been known for a long time that there are persons who have a pronounced faculty of visualization, a capacity for clearly experiencing subjective visual images. Purkinje, Johannes Müller,[1] J. Henle, Fechner, Galton, G. H. Meyer, Lazarus and Kandinsky have each described phenomena related closely to this peculiar "inward vision." It has also been frequently mentioned in different forms by many poets [2] and artists and has been referred to in mystic literature. The otologist Urbantschnitsch [3] studied these subjective images under varying experimental conditions. He made a distinction between optic memory images and optic visualization images. Optic memory images are purely representative; they are not actually seen. Optic visualization images, on the other hand, can be visualized subjectively if the eyes are covered or closed. The latter are distinguished from optical after-images by the fact that they need not follow immediately on actual visual sensations and can be produced voluntarily and by thought associations.

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1. Müller, Johannes: Ueber die phantastischen Gesichtserscheinungen, Klassiker der Medizine, ed. by Sudhoff, Leipzig, Johann Ambrosius Barth, 1927.

2. Kroh, O.: Eidetiker unter deutschen Dichtern, Ztschr. f. Psychol. **85**:118, 1920.

3. Urbantschnitsch, V.: Ueber subjektive optische Anschauungsbilder, Vienna, Franz Deuticke, 1907; Ueber subjektive Hörerscheinungen und subjektive optische Anschauungsbilder, Vienna, Franz Deuticke, 1908; Ueber unbewusste Gesichtseindrücke und deren Auftreten im subjektiven optischen Anschauungsbilde, Ztschr. f. d. ges. Neurol. u. Psychiat. **41**:170, 1918.

Jodl,[4] in 1908, pointed out that these phenomena might possibly explain certain extraordinary performances of memory. E. R. Jaensch and his school, and other later investigators,[5] have systematically studied these visual images. E. R. Jaensch spoke of them as eidetic images (from εἶδος, image—same root as ἰδέα, idea). The eidetic image is midway between an after-image and a representation image, a relationship which one may indicate as follows:

Perception of after-images	Eidetic images	Representation or memory images

The experience of an after-image is the only known instance in which there is "seeing" without an object present (Jaensch). It is believed today that there are transitions from after-images to representation images and that these transition forms correspond to an originally present type of vision which is less clearly differentiated into peripheral and central, or vision and visualization. This undifferentiated form of vision is eidetic imagery. That this transition from after-images to representation images is not only a logical postulate but a factual relationship seems to be shown by the claimed fact that eidetic children who are ignorant of the qualities of negative after-images may produce a negative after-image from a colored object, such as a red square, which is not exposed to them, but which they are asked to imagine (representation after-image).

It was formerly believed that eidetic images occurred only in exceptional and distinctly pathologic persons. The researches of the Jaensch school have shown that they are much more frequent. There are persons, not otherwise conspicuous, who have the faculty of vivid life-like visualization; that is, the production of eidetic images. Such

4. Jodl, Friedrich: Lehrbuch der Psychologie, ed. 3, Berlin, Cotta, 1908, vol. 1, p. 150.

5. Jaensch, E. R.: Die Eidetik und die typologische Forschungsmethode, Leipzig, Quelle and Meyer, 1925. Jaensch, E. R., and Jaensch, W.: The Practical Task and the Importance of Psychological and Psychophysical Researches Among Juveniles, Mitteilungen des Marburger Universitätsbundes, English reprint. (This treatise is a good introduction to the subject of eidetic images.) Jaensch, E. R.: Zur Methodik experimentellen Untersuchungen an optischen Anschauungsbildern, Ztschr. f. Psychol. **85**:37, 1920. Jaensch, E. R., and collaborators: Ueber den Aufbau der Wahrnehmungswelt und die Grundlagen der menschlichen Erkenntnis, ed. 2, Leipzig, Johann Ambrosius Barth, 1927. Jaensch, E. R.: Die typologische Methode in der Psychologie und ihre Bedeutung für die Nervenheilkunde, Deutsche Ztschr. f. Nervenh. **88**:193, 1926; Ueber Methoden der psychologischen Typenforschung (Zur Auseinandersetzung mit G. Heymans), Ztschr. f. Psychol. **108**;1, 1928. Klüver, H.: Description of Eidetic Phenomena, Psychol. Bull. **22**:331, 1925; An Experimental Study of the Eidetic Type, Genetic Psychol, Monogr., 1926, vol. 1, p. 71.

persons are spoken of as eidetic types. Kroh,[6] in 1917, made the important discovery that eidetic images are much more frequent in children than in adults. The faculty of experiencing eidetic images, the eidetic disposition, may exist in childhood and disappear later, or it may persist throughout life.

The existence of an eidetic disposition can be demonstrated by a few relatively simple tests, although for comparative investigations and finer differentiations more complicated experimental conditions are necessary. To test the eidetic disposition of a person, a red square of 5 cm. is exposed against a gray background for fifteen seconds. Usually a negative after-image, which is of the complementary color, appears for a few moments, reappears briefly a few times and finally disappears. In this simple test persons with or without the eidetic disposition show differences in the following four factors: (1) duration of the after-image; (2) course of the appearance of the after-image, either fluctuating or continuous; (3) color, namely, original or complementary color and (4) size of the after-image.

In the eidetic person, the after-images (1) last longer; (2) tend to be continuous and not fluctuating; (3) may have the original color, and (4) may show variations in size which are important for precise investigations, but are more difficult to demonstrate.

Next, instead of a red square, a silhouette representing some simple scene with a few objects is exposed against the gray background. The subject to be tested fixates the silhouette for fifteen seconds. The silhouette is then removed, and he continues to fixate the gray background and describes what he sees. The same test can then be repeated, with the patient closing his eyes and describing what he sees in that way, instead of continuing to fixate on the gray background. The test is then varied by exposing the silhouette for two minutes. The subject, instead of fixating it closely, looks at it leisurely, going over the whole outline and the details in any way he pleases. Then he looks at the gray background and describes what he sees. He is again asked to tell what he sees, after this inspection, when he closes his eyes instead of continuing to look at the gray background.

Depending apparently on individual differences and also on circumstances, eidetic images may be more easily produced sometimes with closed eyes, sometimes with continuation of the fixation (or inspection) of the background. Three types of differences elicited by the tests

6. Kroh, O.: Subjektive Anschauungsbilder bei Jugendlichen, Göttingen, Vanderhoeck and Ruprecht, 1922; Eidetik und Medizin, Deutsche med. Wchnschr. **55**: 600, 1929; Die eidetische Anlage bei Jugendlichen, Ztschr. f. Kinderforsch. **29**:63, 1924.

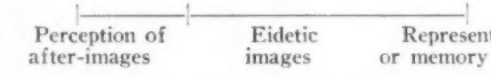
described are of importance: (1) the degree of realization of eidetic images; (2) the color in which the images appear, and (3) the fact that the images appear more easily and intensely after fixation of the object or that they appear more easily after a leisurely inspection.

The following degrees or gradations can be distinguished after fixation of an object: *0*, nothing appears, or perhaps only a border, a fleeting shadow or some spots; *I*, a few details, faintly recognized; *II*, a few distinct details are seen; *III*, the majority of the details of the exposed object are seen, but not distinctly and *IV*, all details are seen distinctly in sharp outline; there is a complete clear reproduction of the stimulus object.

In eidetic phenomena aroused without fixation the following gradations occur: *0*, only an after-image of short duration can be produced; *I*, no production of eidetic image is possible, but eidetic capacity can be inferred from special characteristics of the after-image; *II*, faint eidetic images occur, but only of simple objects (homogeneously colored square) and not of more complicated objects; *III*, weak or fairly clear eidetic images of simple objects; a few details of complicated objects; *IV*, clear eidetic images of complicated objects and *V*, clear and complete images with full details.

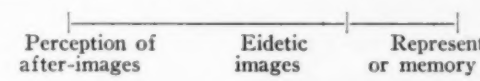
The color of the images is expressed as an index added to the number expressing the degree. Index 1 stands for the eidetic images in the complementary color (white when a black silhouette is used; index 2 for eidetic images which are gray (intermediate) in color and index 3 for eidetic images in the original color. The highest possible degree of eidetic imagery with fixation would therefore be expressed as *IV*.[3]

A difference to which much significance is attributed consists in the question of when the eidetic images occur—whether after briefly fixating the object or after looking at it longer. Two types of eidetic disposition, two eidetic types, are distinguished according to this criterion. In the one type, the eidetic images appear after fixation; they tend to be more rigid or motionless and to have the complementary color. That is to say, in this type (the T-type of Jaensch) the eidetic image is nearer the form of an after-image, a relationship which one may express thus:



In the second type, the eidetic phenomena are produced after looking at the object slowly and at length. The images in this type (B-type of Jaensch) tend to be not rigid, but fluctuating, moving and easily influenced by associations. They appear in the original color. These

eidetic phenomena resemble not an after-image, but a representation image, a memory picture, which relationship may be represented thus:



Schilder,[7] who has himself an eidetic disposition, gave a description of his own eidetic visualization, which corresponds to this second type (B-type). The eidetic images appear in fragments. One half of the picture that was exposed may be almost completely suppressed. The eidetic image does not reproduce the exposed picture in its exact arrangement. There are many displacements and shiftings in space. These displacements may involve either the color or the form or both. Whole figures may be turned around or displaced in space. Color frequently loses its character as "surface color," appears more loose and diffused in space, and becomes a "film color" (as if seen through a diaphragm). The displaced material can be elaborated freely and merged together. Fragmentations of things that belong together may occur. The possibility of transformation of movements is especially marked. A movement may be transferred from something seen in movement to something motionless, or two movements may be exchanged with each other. Frequently the movement appears in these transformations as something strange and unreal. It is a movement per se, to which no natural movement corresponds.

Phenomena similar to eidetic images can occur during fatigue, fasting, emotional states, religious ecstacies and during pregnancy.[8]

The observation that eidetic phenomena are more frequent in childhood seems to have been verified by different observers. But the actual frequency of the incidence of the eidetic disposition and the age at which it is most common seem to be still controversial subjects. One of the difficulties of arriving at a correct estimation of the frequency is that different observers apparently delimit the eidetic disposition differently. Some include "weak eidetic disposition"; others include a latent disposition which they believe to exist. This can be demonstrated by special investigations of after-images and representation images; e. g., the influence of the posture of the head, the influence of the background and the relationship to real objects.[9] It is also claimed by W. Jaensch that by doses of calcium the eidetic pictures of the fixation type can be made to disappear, and the eidetic phenomena of the other type, in which the pictures are more easily produced by

7. Schilder, P.: Psychoanalyse und Eidetik, Ztschr. f. Sexualwissensch. **13**: 56, 1926.

8. Kroh (footnote 6, second reference).

9. Gottheil, E.: Ueber das latente Sinnengedächtnis der Jugendlichen und seine Aufdeckung, Ztschr. f. Psychol. **87**:73, 1921.

leisurely inspection, therefore activated—in this way demonstrating a latent eidetic disposition. This has not been confirmed by later observers. A latent eidetic disposition is also assumed when there are any deviations from the so-called Emmert's law, according to which the size of the after-image grows proportionally to the distance of the eye of the observer from the projection screen. Henning [10] found that there are persons who can produce eidetic images spontaneously, but in whom the Jaensch tests are negative. According to him, therefore, the frequency of eidetic pictures is greater than the Jaensch figures would indicate. There also seems to be a difference in the frequency of the eidetic disposition in different localities. According to Kroh, about 60 per cent of children in Marburg between 10 and 15 years show the eidetic disposition. Allport [11] found only about 50 per cent in Cambridge (England) in children aged 11. Jaensch stated the belief that rudiments of the eidetic disposition exist in almost all children, while pronounced cases occur in 40 per cent. In extensive researches in Hamburg, Bonte [12] found that about one third of the children examined showed eidetic faculty. The greatest frequency was shortly before puberty. According to Rössler,[13] the highest frequency of the eidetic disposition is at the age of 6 (age of beginning school) and it decreases with increasing age. Liefmann [14] also found the greatest frequency (49 per cent) in children aged 6. Several investigators found that the frequency of the incidence of the eidetic disposition is greater in girls than in boys, although this preponderance does not seem to be well established. After adolescence the eidetic disposition disappears. Only relatively few persons are said to carry into adult life the ability to produce eidetic images.

The difference in degree of the eidetic dispositions is considered less important than the difference in their nature. As indicated before, two types can be distinguished: those in whom the pictures are akin to after-images and those in whom they have characteristics nearer to

10. Henning, H.: Starre eidetische Klang- und Schmerzbilder und die eidetische Konstellation, Ztschr. f. Psychol. **92**:137, 1923.

11. Allport, Gordon W.: Eidetic Imagery, Brit. J. Psychol. **15**:99, 1924.

12. Bonte, Theodør: Die personale Bedeutung der eidetischen Anlage unter besonderer Berücksichtigung ihrer Beziehung zum intellektuellen Leben, Ztschr. f. ang. Psychol. **13**:1, 1928.

13. Rössler, Fritz: Verbreitung und Erscheinungsweise subjektiver optischer Anschauungsbilder bei Knaben und Mädchen im Alter von 6-10 Jahren, Ztschr. f. ang. Psychol. **13**:197, 1928.

14. Liefmann, E.: Untersuchungen über die eidetische Veranlagung von Schülerinnen einer höheren Mädchenschule, Ztschr. f. ang. Psychol. **13**:111, 1928; Die eidetische Anlage und ihre Bedeutung für die Konstitutionstypologie, Ztschr. f. d. ges. Neurol. u. Psychiat. **116**:537, 1928. (This is one of the best papers on the subject.)

representation images. On the basis of this difference a whole system of "psychophysical types" has been built. W. Jaensch,[15] a physician and a brother of the psychologist E. R. Jaensch, has developed the theory that there are two constitutional types which show characteristic differences, both in the mental and in the physical sphere. The type the eidetic phenomena of which are more rigid and more easily elicited by fixation he calls the tetanoid type (T-type), assuming a relationship to tetany. The opposite type, with the fluctuating and easily influenced eidetic images, which are more readily elicited by looking at a picture leisurely, he calls the Basedow type (B-type). The T-types are described as showing an overexcitability to mechanical and galvanic stimuli; they are vagotonic; their eidetic images can be made to disappear by giving them calcium, which has no influence on the B-type. Their vegetative nervous system is more sensitive to somatic stimuli. W. Jaensch speaks of them as physiovegetative.

The B-type is characterized by so-called "stigmatization of the vegetative nervous system." This term is used by von Bergmann [16] for a group of individuals with mild hyperthyroid symptoms: tendency to perspiration, elevation of temperature, tachycardia, glistening eyes, slender and asthenic body build, lability of affect, and mild degree of enlargement of the thyroid. The biologic test of Reid Hunt is usually positive. According to Bergmann, this "vegetative stigmatization" is a constitutional attribute, but he uses the term constitution not in the sense of hereditary constitution but as traits acquired during life. Just as the eidetic images are easily influenced by thought associations in the B-type, so their whole vegetative system is more sensitive to psychic stimuli. W. Jaensch speaks of psychophysical interpenetration and a psychovegetative tendency. B-types are vagotonic and sympathicotonic.

Eidetic phenomena are not confined to vision. They occur in all special senses. Henning [17] claimed that in the sphere of the lower senses

15. Jaensch, W.: Ueber Wechselbeziehungen von optischen cerebralen und somatischen Stigmen bei Konstitutionstypen, Ztschr. f. d. ges. Neurol. u. Psychiat. **59**:104, 1920; Ueber die Verbreitung eidetischer Phänomene und der ihnen zu Grunde liegenden psychophysischen Konstitutionstypen (Reaktionstypen), Klin. Wchnschr. **5**:406, 1926. Jaensch, E. R., and Jaensch, W.: Einfache psychische Vorgänge und somatische Korrelationserscheinungen nach sportlicher Leistung und im Training, Sitzungsber. d. Gesellsch. z. Beförderung der ges. Naturwissensch. zu Marburg, February, 1928, vol. 11, no. 2 (Reprint). Jaensch, W.: Grundzüge einer Physiologie und Klinik der psychophysischen Persönlichkeit, Berlin, Julius Springer, 1926.

16. Von Bergmann, G.: Die „vegetativ Stigmatisierten," Med. Klin. **24**:844, 1928; Die vegetativ Stigmatisierten, Ztschr. f. klin. Med. **108**:90, 1928.

17. Henning, H.: Der Geruch, Leipzig, Johann Ambrosius Barth, 1924; Die neuentdeckte Erlebnisklasse der Eidetik, die Urbilder und der Konstitutionstyp, Deutsche Ztschr. f. Nervenh. **81**:180, 1924.

all reproductions of experiences are eidetic. For the sense of touch this has been contradicted by Katz.[18]

Whether there is a relationship between eidetic phenomena and special forms of organization of capillaries demonstrable by capillary microscopy—as is claimed by W. Jaensch—does not seem well established.[19]

The study of eidetic images has been stimulating in experimental psychology, and there is a large literature which has been well discussed by Klüver.[20] The relationships with psychopathology are of great current interest. It is important to realize that the whole subject of eidetic phenomena is still in a developing, not very clear stage. Comprehensive criticisms dealing with both experimental technic and conclusions have been published by Koffka,[21] Allport [11] and Kiesow.[22] The claim of the existence of psychophysical types, the T-type and the B-type, seems to be one of the weakest points. Liefmann,[23] in careful investigations, has not found any relationship between somatic constitutional types and eidetic disposition. Such a relationship is also denied by Fischer.[24]

Of particular interest for psychopathology is the relation between eidetic phenomena and hallucinations. The whole subject of hallucinations is at present in a stage of transition which makes it difficult to point to concrete relationships between experimental psychologic and psychopathologic investigations. Quercy [25] stated the belief that hallucinatory experiences may originate from preoccupation with afterimages. Scharnke [26] pointed to the great complexity and multiformity

18. Katz, D.: Der Aufbau der Tastwelt, Ztschr. f. Psychol. Ergänzungsband, 1925, vol. 11.

19. Suckow, Hans: Die bisherigen Ergebnisse der Capillarmikroskopie für die Klinik, Nervenarzt **1**:300 and 357, 1928. Powdermaker, F.: Capillary Forms in Relation to Certain Problems in Development, Arch. Neurol. & Psychiat. **22**:1207 (Dec.) 1929. Jaensch, W.; Wittneben, W.; Hoepfner, T.; von Leupoldt, C., and Gundermann, O.: Die Hautkapillarmikroskopie, Halle, Marhold, 1929.

20. Klüver, H.: Studies on the Eidetic Type and on Eidetic Imagery, Psychol. Bull. **25**:69, 1928.

21. Koffka, K.: Ueber die Untersuchungen an den sogennanten optischen Anschauungsbildern, Psychol. Forsch. **3**:124, 1923.

22. Kiesow, F.: Zur Kritik der Eidetik, Arch. f. d. ges. Neurol. u. Psychiat. **53**:447, 1925; Kritische Bemerkungen zur Eidetik, nebst an Eidetikern und Nichteidetikern angestellten Beobachtungen, ibid. **59**:339, 1927.

23. Liefmann (footnote 14, second reference).

24. Fischer, S.: Die Beziehungen der eidetischen Anlage zu körperlichen Merkmalen, Ztschr. f. d. ges. Neurol. u. Psychiat. **109**:680, 1927.

25. Quercy, P.: Recherches sur l'eidétisme, J. de psychol. norm. et path. **23**: 702, 1926; Remarques sur les images consécutives, leur pouvoir hallucinogène, autoobservation, Ann. méd.-psychol. **28**:127, 1928.

26. Scharnke, A.: Ueber Beziehungen zwischen den eidetischen Phänomenen und den Sinnestäuschungen, Sitzgungsb. d. Gesellsch. z. Beförd. d. ges. Naturw. zu Marb. **62**:591, 1927.

of hallucinatory symptoms. He expressed doubt as to whether eidetic phenomena can contribute at present to the understanding of hallucinatory reactions. Jankowska [27] has attempted to use eidetic theories in the interpretation of a case of hallucinosis. The patient was a chronic user of alcohol with an acute hallucinatory delirium. He showed the symptom of "Gedankenlautwerden" (thought echo) which persisted after the other symptoms had ceased. He could hear plainly from the left side his own words; e. g., "I lose my position"; "What will happen to my son?" He showed an eidetic disposition and belonged to the B-type. Jankowska concluded that there is an inner relationship between the symptom of thought echo and eidetic images. The thought echo is a sort of regressive phenomenon in which there is no sharp differentiation between a representation and a perception, just as in childhood, according to Jaensch's hypothesis, there is an undifferentiated stage in which representation and perception are practically unified to become two different phenomena only in later life. Fischer and Welke [28] have observed eidetic pictures with reality character in cases of psychosis and of psychopathic personality. The patient believes that the image he sees is real and tries to find it or to talk to it. Catathymic factors play a part in the development of these sensory experiences. It is interesting that all these patients were young, between the ages of 8 and 18. In a study of fifty schizophrenic patients, Miskolczy and Schultz (Eidetik und Schizophrenie, Monatschr. f. Psychiat. u. Neurol. **72**: 354, 1929) found the eidetic disposition more frequently among hallucinating patients than among those who did not hallucinate.

The eidetic disposition is also regarded as exerting an influence on memory and intelligence. Busse [29] considered after-images, eidetic images and representation or memory images as three stages or levels of memory, the after images being the lowest, the representation images the highest stage. It is apparently possible for the eidetic disposition to enable a person to give extraordinary memory performances. Jaensch and Mehmel [30] have published the report of a case of a feebleminded young man who could name quickly the day of the week of any past or future date. It was found that he could visualize the figures on the calendar vividly. He saw the date-figures, with each Saturday underscored. It does not seem to be clear to what extent an eidetic

27. Jankowska, H.: Ein Fall von Gedankenecho oder Gedankenlautwerden, abstr. by Higier, Nervenarzt **2**:118, 1929.

28. Fischer, S., and Welke, W.: Eine neue Art von Trugwahrnehmungen, Arch. f. Psychiat. u. Nervenh. **76**:143, 1926.

29. Busse, P.: Ueber die Gedächtnisstufen und ihre Beziehung zum Aufbau der Wahrnehmungswelt, Ztschr. f. Psychol. **84**:1, 1920.

30. Jaensch, E. R., and Mehmel, H.: Gedächtnisleistungen eines schwachsinnigen Eidetikers, Psychiat.-neurol. Wchnschr. **30**:101, 1928.

disposition may help in retention and reproduction. But it is interesting in this connection that in the production of eidetic images there seems to be operative a process of "selection."[31] That is to say, a person who is capable of producing eidetic images reacts differently to different contents. If, in the example of Jaensch, he is fond of botany and less interested in zoology, though a student of both, he may produce clear eidetic images of botanic subjects but only vague ones of zoologic subjects. Moreover, emotional preferences influence these optical phenomena. Jaensch expressed the belief that it can be shown that there may be a difference in some persons according to whether they attempt to visualize eidetically an object of some sort, on the one hand, or merely a geometrical figure such as a circle or square, on the other. This preference for an object he speaks of as an "ontotropic" tendency.

Several investigators have assumed a close relationship between intelligence and eidetic disposition. Zillig[32] and Kirek,[33] for example, stated that there is an inverse relationship between pronounced eidetic faculty and good intellectual endowment. Flournoy[34] has described the case of a feebleminded young woman who could visualize absent people with such great vividness that she had difficulty in distinguishing between her representations and her actual perceptions. Her statements were easily influenced by the form in which questions were put to her. Flournoy believed that his observation confirms the view held by Jaensch that primitively a mental representation is easily confused with a perception. Bonte[12] found no correlation between B- and T-types and intelligence. They occurred at all intellectual levels. The performance of children in different mental tests varied not according to their eidetic disposition but according to their general intellectual endowment. Intelligent eidetic persons may make use of their eidetic faculties, but in persons "with weak determination" the eidetic disposition may become a relative disadvantage. There is said to be a strong correlation between the eidetic B-type disposition and susceptibility to suggestion. The eidetic disposition has no influence on abstract thinking, but it may influence the performance of mental tests into which enter attention for visual objects, power of observation for concrete objects, visual memory or the reception and elaboration of visual psychic contents in general. Rössler[13] also found no correla-

31. Jaensch, E. R.: Ueber psychische Selektion, Ztschr. f. Psychol. **98**:129, 1926.

32. Zillig, M.: Ueber eidetische Anlage und Intelligenz, Fortschr. d. Psychol. **5**:293, 1922.

33. Kirek, H.: Die Bedeutung der sensoriellen Veranlagung für die Bildung von Objektvorstellungen, insbesondere bei Eidetikern, Ztschr. f. d. Erforsch. Behandl. u. d. jugendl. Schwachsinns **46**:121, 1926.

34. Flournoy, H.: Eidétisme chez une débile, Arch. de Psychol. **20**:73, 1927.

tion between eidetic disposition and intelligence. He expressed the belief that the eidetic faculty may be utilized to advantage pedagogically in certain fields—as in geography, in which the eidetic child can visualize a map. Carmichael [35] has drawn attention to the fact that the reactions of a child to certain parts of the Binet-Simon test may indicate the existence of an eidetic disposition.

The study of eidetic images also throws light on problems of primitive thought. This is of significance for psychiatry on account of the analogies with psychopathologic phenomena, especially in schizophrenia. Jaensch [36] stated the belief that the eidetic disposition constitutes not only an ontogenetic phase, but also a stage in phylogenetic development, and that primitive races generally tend more to eidetic experiences. Storch [37] has pointed out the significance of eidetic phenomena for the understanding of peculiarities of schizophrenic thinking.

The significance of eidetic phenomena for psychiatry is not restricted to formal considerations. The content of eidetic visualization may give indications of deep-lying trends. Kroh [38] expressed the belief that the content of eidetic images may give an important clue to the understanding of the personality of children and to the diagnosis of mental conflicts and abnormal trends. Schilder [7] has pointed out the analogy between the characteristics and changes of the details of the eidetic visualization of an object or scene, and the "pictures" which emerge during the psychoanalysis of patients with eidetic disposition of the B-type. "The eidetic images follow instinctive tendencies. These strive to repress that which is disagreeable to the individual." Schilder expressed the belief that there is a close connection between the ideas expressed in Jaensch's paper on "psychic selection" [31] and the psychoanalytic theory. What Jaensch calls "kalotropic tendency"—that is, the tendency to visualize pleasant objects more clearly and completely, while unpleasant ones are modified or do not appear at all—is the same as Freud's repression of disagreeable contents. Schilder contradicts Jaensch's view that the freudian mechanisms apply only to eidetic persons of the B-type. Repression, displacement and condensation are operative not only in the eidetic sphere, but also in the sphere of representation and thinking, and in psychoses in the sphere of perception. Jelliffe [39] agrees with Kroh that the study of the content of eidetic

35. Carmichael, Leonard: Eidetic Imagery and the Binet Test, J. Educ. Psychol. **16**:251, 1925.

36. Jaensch, E. R.: Die Völkerkunde und der eidetische Tatsachenkreis, Ztschr. f. Psychol. **91**:88, 1923.

37. Storch, A.: Erlebnesanalyse und Sprachwissenschaft, Ztschr. f. Psychol. **94**:146, 1924.

38. Kroh (footnote 6, first reference).

39. Jelliffe, Smith Ely: On Eidetic Psychology and Psychiatric Problems, M. J. & Rec. **128**:80, 1928.

images may yield important information for psychotherapy and prophylaxis: "I feel certain that many a life catastrophe may be averted when these (eidetic) studies become more formalized and made practically available for us, as psychiatrists. Herein we may learn much about objective testing of repressions which may contain most outstanding danger signals for aftercoming development. Thus may there not be established a liaison between objective testing and psychoanalytic study of children which will be of far-reaching service in that process which we hazily designate as 'socialization' of the libido." Krassnuschkin [40] stated the belief that the distinction of eidetic types in the sense of W. Jaensch is important for the understanding of psychogenic disorders. The B-type furnishes the soil for dream-like delirious states, the T-type for emotional outbursts and stuporous conditions. It seems almost unnecessary to add that for a pupil of Adler the eidetic disposition is due to "organ inferiorities" in the sphere of the optic apparatus, compensated especially in puberty with its striving and training for adulthood (Friedmann [41]).

Schilder has drawn attention to the similarity between the visualization of eidetic persons and the descriptions of perceptions of patients with "mind blindness." Other problems of neuropsychiatric interest which have connections with eidetic psychology are micropsia and macropsia,[42] the influence of stimulation of the labyrinth on perception and visualization [43] and experimental psychoses produced by mescal.[44] The claim of W. Jaensch that eidetic imagery can be induced by mescal does not seem to have been confirmed.

It is difficult to summarize the significance of eidetic phenomena from the psychopathologic point of view. There is a large literature on the subject. It consists partly of excellent experimental work, but a great deal is devoted to far-reaching conclusions and vast generalizations. After all, the fundamental problem arises whether

40. Krassnuschkin, E. A.: Zur Frage der Einteilung der Psychogenie, Ztschr. f. d. ges. Neurol. u. Psychiat. **115**:615, 1928.

41. Friedmann, A.: Eidetik und Individualpsychologie, Ztschr. f. Individualpsychol. **5**:196, 1927.

42. Neuhaus, W.: Makropsie und Mikropsie bei Basedowoiden. Ein Beitrag zur Konstitutionsforschung und Entwicklungsgeschichte, Ztschr. f. d. ges. Neurol. u. Psychiat. **105**:257, 1926.

43. Bibring-Lehner, G.: Ueber die Beeinflussung eidetischer Phänomene durch labyrinthäre Reizung, Ztschr. f. d. ges. Neurol. u. Psychiat. **112**:496, 1928.

44. Klüver, H.: Mescal Visions and Eidetic Vision, Am. J. Psychol. **37**:502, 1926. Beringer, K.: Der Meskalinrausch, Berlin, Julius Springer, 1927. Mayer-Gross, W., and Stein, H.: Ueber einige Abänderungen der Sinnestätigkeit im Meskalinrausch, Ztschr. f. d. ges. Neurol. u. Psychiat. **105**:257, 1926. Quercy, P.: Auto-observation d'hallucinations visuelles, J. de psychol. norm. et path. **24**: 520, 1927.

eidetic phenomena can play a leading rôle in personality development, such as is ascribed to them, or whether they are not of more incidental significance. It seems exceedingly doubtful whether the study of these psychovisual phenomena can be made the basis of whole systems of psychology or of constitutional types. There are as yet few concrete instances in which these investigations contribute definitely to psychiatric problems. But even from these beginnings it would seem that the study of eidetic phenomena has great significance for psychopathology. Here are systematic experimental investigations of phenomena which lie between the physiologic and the psychologic sphere. One of the most successful chapters of experimental psychology has been the physiology of special senses (Sinnesphysiologie) in which a vast amount of painstaking work has been done over a period of many years—and with much less speculative ado than in other branches of psychology. That a bridge is found to connect these studies with broader problems of personality constitutes alone a noteworthy advance promising interesting future developments. Even though the relationships are as yet not clear, the study of hallucinations cannot but profit eventually by experimental investigations such as those stimulated by researches on eidetic phenomena. But one of the chief results which the study of eidetic phenomena has contributed so far to psychopathology is the demonstration that, at least in the psychosensory sphere, a veritable "metamorphosis" takes place in human development between childhood and maturity.

Abstracts from Current Literature

A Clinicopathologic Study of Disease of the Brain in Early Childhood with a Predilection for the Striatal System. E. Fünfgeld, J. f. Psychol. u. Neurol. **40**:85 (Dec.) 1929.

The patient whose case is reported was born in 1901; labor was difficult and development retarded; he did not walk until the age of 1½ years. Although he apparently carried out commands, recognized persons and objects, and was very playful, he learned to speak only a few words. At the age of 5 months he began to have epileptiform seizures which appeared at first daily but later every few weeks. At the age of 15 years he gradually developed a tremor of all four extremities with psychic changes characterized by increasing irritability and loss of interest. At the age of 22, the limbs became progressively rigid so that at 24 he was unable to get about at all; his general condition became markedly deteriorated.

On admission to the clinic he showed: expressionless facies, no tendency to spontaneous motility and periodic stereotyped jerking of the upper lip. Only the simplest commands could be executed; general asthenia with rigidity of the muscles of the neck and of the upper extremities was noted (more marked in the proximal portions of the arm). There was slight tremor in the muscles of the shoulders increasing in severity distalward and on intention. Rigidity was more marked in the lower limbs, especially on the right side; the right big toe was in dorsal flexion; the patient could walk only with assistance and then the body would be bent forward, very small steps would be taken, and the trunk and head held in flexion and to the right. There were loss of normal associated movements, occasional tendency to propulsion and facial grimacing, and complete mutism. The pupils reacted to light promptly but not in convergence; ocular movements could not be tested. There were present right sided facial weakness; very lively arm reflexes; markedly diminished hand grip and abdominal reflexes; very lively knee jerks, with occasional ankle clonus and no Babinski sign. While testing for sensation, the arms and legs would develop a very coarse tremor; this would also occur during passive motion of the limbs.

Course.—During the patient's stay at the clinic no convulsions were observed. The neurologic status remained unchanged but the general condition became gradually worse and the patient died early in February, 1926.

Necropsy.—Except for the pallidum, which appeared perhaps darker than normal, the brain showed nothing abnormal macroscopically. The cerebral vessels were thin. The brain weighed 1,350 Gm. The other noteworthy necropsy observations were: tuberculous pleurisy, fibroid phthisis, a small heart, cloudy swelling of the parenchymatous organs and fatty degeneration and infiltration of the liver.

Microscopic Examination of the Brain.—Nissl Stain: The cerebral cortex was somewhat richer in glia nuclei than normal; this applied to all layers of the cortex the structure of which, except for occasional shrinkage of the pyramidal cells in the third and fifth layers, was normal. The ganglion and glia cells contained an unusually large amount of fat. The vessel walls were in some areas somewhat thickened with collections of cells unusually rich in nuclei; the cells of the adventitial coat contained moderate amounts of lipoid. The pia was intact. The Betz cells were well preserved.

Myelin Fiber Preparations (Spielmeyer-Kulschitsky-Pal): In all fields examined (10, 8, 9, 4, 1, 2, 3, 20, 21, 22, 41, 42, 37, 5, 7, 39, 17, 18, 19) there was a definite diminution in the ground fiber network, especially in the deeper layers, whereas in the more superficial layers there was an abnormal discoloration of the myelin sheaths, particularly the delicate fibers in the fourth layer. Although with the Bielschowsky stain the axis cylinder network was well preserved, there

was a slight but definite fibrous gliosis with glial fibers extending down the fourth and beginning to invade the fifth layers. Except for a diminution of the ground fiber network in Ammon's horn, this structure appeared normal.

The ganglion cells in the caudatum and putamen contained considerable quantities of lipoid; otherwise they were normal. There was, however, in these two ganglia a definite increase in glia nuclei. The thalamus was small and the ganglion cells and glia nuclei were strikingly dense, especially in the upper lateral and median nuclei. The fornix and anterior commissure appeared narrower than normal and were very rich in glia nuclei.

The pallidum also contained an unusually large number of glia cells. In its ventral third and, to a lesser extent, in the part adjacent to the putamen there were large quantitives of desposits symmetrically situated on the two sides; these deposits were of four varieties: 1. Small, deeply stained ball-shaped masses, some of which surrounded the vessels and others were situated diffusely in the depths of the tissue. As far as one can determine from the different staining reactions they represented precipitates of the tissue fluids. 2. Pigments, which with the Nissl stain appeared partly homogeneous and partly metachromatic; they were situated in the vessel walls and in the glia, and yielded the iron reaction. The quantity of this pigment was relatively slight. 3. The walls of many large veins were impregnated with "pseudocalcium" which stains red with thionine and also gives the iron reaction. 4. Lipoid material which could be seen in fairly large amounts in the vessel walls as well as in the glia.

The ganglion cells of the pallidum were generally free from involvement except that here and there one could see rests of destroyed cells which appeared as small, dark stained structures with ill defined nuclei. Aside from the previously described, deposits, there was no visible increase in the iron content of the tissues.

The appearance of the myelin fibers with myelin sheath stains was practically the same in the caudatum, putamen and pallidum as in the cortex. There was a definite reduction of the axis cylinders in the pallidum, and also a slight increase of fibrous glia in the striatum.

In the myelin sheath preparations the thalamus appeared poor in myelin; the delicate fibers in the upper lateral nuclear regions and in the ventral nucleus were poorly stained, although they did not seem to be diminished in number; on the other hand, there was unquestionably a loss of these delicate nerve fibers in the median nucleus up to the pulvinar and especially in the ventral portions. In the Bielschowsky preparations the thalamus appeared normal, though there was probably a diminution of fibers in some areas between the thalamus and pallidum.

The ansa lenticularis was poorly stained but there did not seem to be an actual diminution of the fibers. The H 2 bundle was abnormally thin but the H 1 appeared normal. The corpus luysii was intact.

In the mamillary body, as well as in the central gray, there was considerable decolorization of the fine delicate myelin fibers although it is difficult to say whether their number was diminished. The fornix and anterior commissure were almost entirely decolorized; this area was definitely smaller and also showed a fibrous gliosis.

The substantia nigra showed a remarkable lack of melanin in its ganglion cells which appeared smaller than normal, although they were not diminished in number and were overloaded with lipoid material. The cells in the reticular zone were intact but the glia in this region showed an enormous amount of lipofuscin. The entire substantia nigra was poor in delicate nerve fibers but the coarse fibers were well preserved. The lateral portion of the tegmentum was definitely smaller and showed a diminution in the number of fibers, but the medial portion did not take the stain well although it could not be definitely stated whether the fibers were diminished in number, nor could it be determined whether there was an actual increase of glia or a definite diminution in the fibrils.

The red nucleus, geniculate bodies, peduncles, corpora quadrigemina and medulla showed no definite pathologic changes, although here and there (ventral

portion of the fibrous capsule of the red nucleus and in the medulla) the myelin fibers did not take the stain very well.

The dentate nucleus was intact, as was the entire cerebellum.

Comment.—The clinical picture in this case can be well correlated with the anatomic observations. The congenital mental defect would seem to be due to the definite diminution of the myelin fibers within the cortex. The disturbances in motility are due to pallidal involvement. It is difficult to determine how much of these disturbances is due to involvement of the substantia nigra, although it is noteworthy that the pallidothalamic fibers are less reduced in number whereas the pallidonigral tracts are much more involved in the pathologic process. Finally, the hyperkinetic phenomena could be attributed to the diminution of the striopallidal fibers which connect the pallidum with the large cells in the striatum, and to the involvement of the striatal fibers themselves.

In contrast to the cases described by O. Fischer, the histologic preparations in this case show nowhere any indication of destruction of the parenchyma of the nerve tissue. The lipoid content in this case does not differ from that usually observed in the case of any person who has succumbed to a chronic infection. The deposits found in the various structures can be indicative only of metabolic changes of a chronic pathologic disorder and cannot be regarded as evidences of a necrobiosis. It may, however, be possible that the definite lack of melanin in the compact zone of the substantia nigra is of some pathogenetic significance. It is well known that in the new-born child the compact zone contains no melanin, and that it is not until the fourth year of life that this pigment gradually begins to develop. In view of this, one is unquestionably dealing in this case with an inhibition of melanin development which, according to the Vogts, may also be the basis for an inhibition of myelinization. Against this theory of failure of myelinization in this case, however, is the clinical fact that the disorders of motility did not appear until the patient had reached his fifteenth year, although there would seem to be no difficulty in attributing the seizures which began in the fifth month of life and the mental defect, which was also noticed very early in life, to defective myelogeny of the cortex. The next question then is, why did the motor disturbances first appear so late? According to Kaes, myelinization of the cerebral cortex, especially of the principal external layer (1-3), is not complete until well after the thirtieth year of life. One may also readily conceive that in the brain stem too some fibers do not become completely myelinated till after the termination of the second decade of life. Finally, the pathologic changes in the case under discussion would seem to point to a disturbance of the "internal structure" of the basal ganglia and especially of the pallidofugal fibers. The author believes that it cannot be definitely excluded that myelinization of the basal ganglia ran its normal course during the early years of life, but, as in the cortex, it became inhibited during later development. He also has no doubt that the pathologic process in this case is closely related to that described by the Vogts as status dysmyelinatus, and that the inability of the tissues to take up the various stains represents merely a slight degree of this condition.

KESCHNER, New York.

A CONTRIBUTION TO THE STUDY OF THE PELLAGROUS DISEASES. W. OPPLER, Ztschr. f. d. ges. Neurol. u. Psychiat. **123**:27, 1929.

The author reviews the literature on pellagra in Germany. Most of the cases have psychotic symptoms. Oppler presents his case as an addition to the picture of the disease and also to show that it is invading Germany more and more.

The patient, a woman, aged 44, was transferred to the psychopathic institute ten days after her admission to the city hospital. She was completely confused and refused food, saying it was poisoned. She was disoriented as to time and place. Becoming suddenly excited, she broke a window. On admission she seemed anxious, kept looking around, and would answer only questions concerning the window she had broken. All that day she continued to be very restless. That night she became quieter and the following day lay in bed,

her face drawn, and repeated "I know nothing." She could still say her name, but not her age or address. Later she said that there were snakes in her bed, then on her body. During the following days she became more and more mute, continuously lay in bed with her face drawn with anxiety, and became frightened whenever any one approached or touched her. She spoke only single words and refused to eat, fearing the food was poisoned.

After a few days she seemed slightly better and answered a few questions more or less sensibly, but she was still disoriented as to time and place and had delusions of persecution. She remained in this comparatively quiet and passive state till the eighth day after admission when she suddenly became violent, attacked the nurse, and would allow no one near her. This wildness lasted two days, during which she had to be isolated and given scopolamine hydrobromide.

From then on she became progressively weaker and remained in bed. A week after the attack of excitement, diarrhea set in and disappeared and reappeared from then on. For another month she lay in this weak apathetic state, occasionally becoming delirious and excited and again lapsing into mutism. At the end of the month, choreiform movements of the arms, hands and later of the face set in. Ten weeks after admission, she had sudden clonic twitching of the face and became pale, the pulse became poor and she died.

The patient had been dehydrated on admission; the tongue was red; the pupils reacted; eye movements were free; there was no nystagmus; the cranial nerves were normal; the abdominal reflexes were absent, the patellar and the achilles reflexes increased. During the course of the illness these reflexes became still more marked; the lower limbs and gait became spastic. On the back of both hands at the time of admission there were barklike thickenings of the skin with scaling. The thickenings were sharply demarcated and parts were pigmented a light brown. The scales were constantly falling off and continuously being more richly replaced. Gradually the condition spread to the palms, and areas appeared on both feet and elbows. On both sides of the nose small cone-shaped areas appeared. The spinal fluid showed nothing of note. A postmortem examination was refused.

The past history given by the daughter was of interest. Because of digestive disturbances which dated back four years, the patient had eaten no meats for two years, living mainly on potatoes and salads, leguminous plants and bread. She drank milk and ate plenty of butter, but never took maize or any food containing cornmeal. She became constantly thinner and weaker. For four or five months before admission to the hospital she noticed that her hands were "peeling," and about three months before, the first psychic changes appeared. She became forgetful, anxious, and developed delusions of persecution. She had no fever or headaches during this time.

The author expressed the belief that there is no doubt of the diagnosis, that the case is one of a genuine pellagrous psychosis. The gastro-intestinal history, the skin changes and onset were typical, but the psychotic symptoms were not. At first there was the paranoid, anxious state, and then the onset of the periods of marked motor excitement. Mainly there was an asthenic, anxious state, at times stupor and negativism, which was interspersed with phases of delirium. The psychosis shows throughout its course great similarity to the cases reported by Bonhoeffer and Chotzen. The neurologic signs were: spasm with increased reflexes, absent abdominal reflexes and later choreiform movements. The mucous membrane irritation was shown by gingivitis, diarrhea and metrorrhagia.

What this patient's original four years' gastric distress was due to, the author cannot say. From this case and his observations of others reported, he believes that any nutritional disturbances in a particularly predisposed person can be the causative factor for the pellagra symptom-complex. What factor renders a person "predisposed" is still unknown. Goldberger believed that the condition is one of avitaminosis, vitamin B being absent. In this case, however, the patient had taken a diet rich in all the vitamins, omitting only meat, which contains no vitamin B. It seems important that the protein intake was mainly of a vegetable

character, milk and eggs supplying the only animal protein. The case reported by Kleiminger showed this same peculiarity of diet, and that author believed that the absence of animal proteins was the reason for the pellagra. Oppler does not go so far, but he thinks that perhaps a one-sided diet, whether lacking in vitamins or in animal proteins, may be the causative factor.

ALPERS, Philadelphia.

NYSTAGMIFORM CLONUS IN AN EPILEPTIC. DIRKS-DILLY, Rev. d'oto-neuro-opht. **7**:341 (May) 1929.

This article presents a report of a patient with symptoms limited almost entirely to nervous crises and vague nystagmiform movements of the eyes. No somatic lesion was discovered, and on the side of the nervous system there was neither motor, sensory or cerebellar disturbance nor any contractures or trembling. The reflexes were a little lively, and the vegetative system presented a degree of disequilibrium.

The patient, aged 22, was almost suffocated at birth by the umbilical cord being wrapped around the neck. Except for nervousness he was well up to the age of 15, when he had some digestive disturbance. At the age of 18, he was in bed for two months with pain in the hip. About this time the nervous crises appeared. His occupation was that of a waiter but though he would fall suddenly, this never happened during service at a table. He was inducted into military service at the age of 22 and the crises became more frequent. The surgeon in the hospital noted that a rigid alimentary regimen suppressed the attacks. A pretended faradic treatment would bring on profound sleep and on waking he would have the feeling of having had a crisis. He said that he had not suffered from syphilis or alcoholism.

Examination revealed a man of medium figure, with no fever, stigmas of degeneracy or evidence of wounds of the head or tongue. Except for the heart action, which was rapid and unstable, all bodily organs were clinically sound. The arterial tension was 15 systolic and 8 diastolic; the Wassermann reaction was negative. The eyes were large and immobile on looking ahead; but when he attempted to follow the finger with the eyes, a crisis came on and was characterized by a clockwise movement of circumduction of the globes. This movement could not be arrested by a command, but stopped when his attention was diverted. The movement was accentuated and accelerated during tests of cerebellar function, compressing the carotids and pinching the cheeks.

The patient was discharged from military service as an epileptic subject. The evidences pointing to organic trouble were: exophthalmos, instability of the pulse, exaggerated reflexes and rapidity of the eye movements. During three months of idleness, the crises were numerous, appearing every three or four days, shortly after eating, with loss of consciousness. Sitting down would avert a crisis. Attacks of abdominal colic appeared, and after that the nervous crises were less severe. He became absentminded and forgetful, and did not respond when spoken to; on coming to himself, he would ask where he was and why he had done a certain thing; if he thought of some one, he would see him as though present; he became irritable and violent-tempered.

An examination of the eyes showed normal visual acuity and eyegrounds. Atropine produced rapid and extreme mydriasis. In the act of aiming a gun the eyes would not remain fixed, but after some circular movement there would be a slight fibrillation of the globes and lids. This occurred also with the eyes closed.

An examination of the ears and vestibular apparatus revealed a slight impairment of hearing, but no vertigo or disturbances of equilibrium. Rotation caused accentuation of the gyratory movement of the eyes but no true nystagmus. Irrigation of the left ear with cold water did not modify the eye movements. The galvanic test did not change the eye movements. A moving picture was made which permitted a detailed study of the movements. The face showed no contractures of the forehead, mouth or cheeks, which probably would have accompanied voluntary movement of the eyes.

A crisis was witnessed. The patient suddenly fell backward without an outcry and with the arms extended. The eyes were open, and the globes moved rapidly in circles. There were rapid jerking movements of the arms and legs, the mouth opened and closed two or three times. The face was white above and violet below. The crisis lasted five minutes and was followed by a sense of fatigue. All of the symptoms described could be simulated, except the color of the face and the rapid circular movements of the eyes. The organic nature of the trouble is supported by the following: the marked effect of atropine, the colics, the nonreaction of the vestibular apparatus, the emotivity of the patient, causing excessive reactions to examination, the exophthalmos and the instability of the pulse. The coloration of the face, the absentmindedness and the modifications of character speak for epilepsy. As to the location of the lesion, the author cannot express an opinion as to whether it was in the basal nuclei, the ocular nuclei or the supranuclear centers. The suggestion is made that infection could be the causative agent, since oculogyric crises have been observed in epidemic encephalitis, and, in the case reported, there was an undetermined illness at the age of 18 which confined the patient to bed for several months — possibility encephalitis.

DENNIS, Colorado Springs, Colo.

DISSEMINATED SCLEROSIS. PATHOLOGICAL AND BIO-CHEMICAL CHANGES PRODUCED BY A "VIRUS" CULTIVATED FROM THE CEREBROSPINAL FLUID. J. A. B. HICKS, F. D. M. HOCKING and JAMES PURVES-STEWART, Lancet **1**:612 (March 22) 1930.

Soon after the fact became established (Chevassut) that a virus was constantly found in the cerebral fluid in patients with disseminated sclerosis, it was decided to study the effect of this virus on animals. A method was devised by Hicks for estimating the strength of the virus and is given in detail in the paper. The vaccine suspensions were treated with a 0.5 per cent phenol-saline solution. Experiments on rabbits, in an attempt to produce an antiserum, gave results indicating that these animals could produce inhibitory substances in about from two to five weeks. It had already been noted that the serum of treated patients was also viricidal. The serum of the experimental monkeys did not develop inhibitory properties. Experiments on rabbits and on the serums of twelve patients with disseminated sclerosis failed to reveal any evidence that fixation of complement occurred.

Experiments were done on seven monkeys in an attempt to produce the lesions of disseminated sclerosis. Both cisternal and intravenous injections of broth cultures were given. In only one case was a really significant result obtained. This monkey received a single intravenous injection of 5 cc. of the culture of the virus. Cultures of the spinal fluid of this monkey made at one, two and six months' intervals were positive for the virus. About nine months following the injection, a transient paresis was observed in the hind limbs of the monkey. There were several similar attacks, and after fourteen months the paresis became general and the animal was killed. Sections of the spinal cord stained by the Marchi method revealed two distinct tracts of system degeneration; one was situated in the left posteromedian column and the other in the direct cerebellar tract on the left. In some of the sections stained with hematoxylin and eosin there seemed to be perivascular cuffing, but the blood vessels were unaffected. One other monkey, who had remained clinically well but whose spinal fluid had yielded positive cultures, showed at autopsy a well marked degeneration at the periphery of the anteromedian column and scattered patches in the lateral columns. The animal experiments are being carried further.

Since one of the characteristic lesions in disseminated sclerosis is a degeneration of the medullary sheath with splitting off of the unsaturated acids of the oleic acid series (hence the black stains with osmic acid), it was decided to investigate the ability of cultures of the virus to split these lipoids in vitro.

Since the reactions would result in the liberation of either free fatty acids or of phosphoric acid the H-ion concentration could be used as an indicator. A fatty emulsion of fresh human brain was made. It was first observed that the virus would grow in this emulsion. Then cerebrospinal fluids from patients with other diseases were incubated in Hartley's broth plus the emulsified brain and the H-ion concentration studied. It always remained well within the limits of experimental error. Thirdly, characteristic curves were obtained of the changes in p_H resulting from the growth of the virus itself on the usual broth-serum medium. With these as controls, a series of cultures were inoculated into the fatty emulsion and daily readings of the p_H taken. Instead of the anticipated increase in acidity it was found that after a short period in which there is a slight increase in acid formation there is a progressive diminution in acidity which tends toward a constant reading. The author (Hocking) concludes that this is due to the fact that the free fatty acids are not primarily split off from the fatty molecule as had been hypothecated, but the nitrogenous base, choline. This is in agreement with Halliburton's discovery of choline in the spinal fluid in cases with nerve degeneration. Ethereal extracts of the fatty medium were made before and after incubation with the virus and the determination of the acid values did not indicate any splitting off of the fatty acids as a result of the activity of the virus.

It may be concluded that "cerebro-spinal fluids containing this particular virus exert a specific hydrolytic action not only on proteins and their disintegration products but also on fatty constituents which occur in nervous tissues."

GINSBURG, New York.

A SPECIFIC VACCINE TREATMENT IN DISSEMINATED SCLEROSIS. JAMES PURVES-STEWART, Lancet **1**:560 (March 15) 1930.

At the request of the author, Miss Chevassut used cultures grown by her from the cerebrospinal fluid of patients with disseminated sclerosis in preparing an autogenous vaccine. Its effects serologically and clinically are reported in a series of 128 cases, seventy of which remained under observation long enough to be carefully followed. In order to eliminate the factor of protein shock, vaccines were prepared in physiologic solution of sodium chloride. The cultures were killed by the addition of 0.5 per cent phenol and were not filtered. The standardization of the vaccine was done by counting under the microscope, the important factor being the number of granules present.

The serum of treated patients has been observed to acquire viricidal properties, which is in harmony with the consensus of opinion that filtrable viruses have the power of stimulating the production of antibodies. This property is highly specific. In 27 of 32 cases it was found that when the serum acquires such a property it is inhibitory only to the virus cultured from the patient's own cerebrospinal fluid.

Purves-Stewart emphasizes the difficulty of estimating the true value of treatment in disseminated sclerosis because of its tendency to undergo spontaneous remissions. However, in these cases the clinical arrest was coincident with the disappearance of the organism from the spinal fluid. In spontaneous remissions the spinal fluid shows the usual positive gold curve and globulin reaction and contains the organism; in a true therapeutic remission as a result of vaccine treatment the fluid loses its pathologic reactions and becomes sterile.

The author quotes four illustrative cases to emphasize the value of the culture as a diagnostic method as well as to point out the changes under treatment. The clinical results may be summarized as follows: of ten patients with early cases, nine improved and one remained in statu quo; of twenty-seven patients with moderate cases, twenty-two improved and five were not improved; of thirty-three patients with advanced cases, nine improved and twenty-four were not improved.

Serologic observations yielded the following results: of ten patients with early cases, four developed a negative culture and eight showed improvement in the gold and globulin reactions; of twenty-seven patients with moderate cases, four developed negative culture, and twenty-one showed improvement in the gold and globulin reactions; of thirty-three with advanced cases, in none did the culture become negative, while twenty showed improvement in the gold and globulin reactions. Purves-Stewart emphasizes the fact that the most that can be hoped for in any kind of treatment is to arrest the malady at the stage it has already reached; restoration of degenerated nerve tissue to normal is not to be expected.

GINSBURG, New York.

ANATOMY AND PATHOLOGIC PHYSIOLOGY OF SYDENHAM'S CHOREA. J. LHERMITTE and P. PAGNIEZ, Encéphale **25**:24 (Jan.) 1930.

A review of the literature shows that a variable pathologic base is described in chorea. In 1920, Mery and Babonneix reported an observation of extensive pathologic changes of the cerebral cortex and of the basal ganglia, consisting of vascular infiltrations and cellular degenerations. In 1921, Henri Claude published observations of distention of the vascular sheaths, without infiltration, and of cellular degeneration in the basal ganglia. Delcourt and Sand reported a case with maximum involvement of the basal region, becoming lessened as one approached the cortex, bulb and cerebellum. Tinel, in 1914, had reported similar disturbances in the ganglia and in the frontorolandic area. Marie and Tretiakoff, in 1920, published accounts of marked vasodilatation and infiltration of the adventitial sheaths with mononuclear cells, lymphocytes, plasmacytes and myelophages, both in the cortex and in basal regions. They also discovered inflammatory reactions of the locus niger. Similar studies were made by Harvier and Levaditi; also by Harvier and Decour in a man, aged 86, succumbing to acute chorea and showing at autopsy vascular thrombosis with perivascular infiltration in the cortex. Slauk found intense lesions of the caudate, the thalamus, nucleus ruber, corpus Luysi and the perisylvian region. These were agglomerations of neuroglia.

Then follows a report of the case of a woman, aged 19, whose illness commenced early in November and progressed as a typical chorea to death in December. Grossly, there was evident congestion of the meninges. Microscopically, the examination was made in a most detailed way and yielded results which are summarized as follows: vasodilatation, vascular rupture with capillary hemorrhage, distention of the adventitial sheaths by an albuminous exudation and cellular changes—tigrolysis and karyolysis, with the gradual reduction of the cytoplasm and the final destruction of the cell. As to topography, the lesions were predominant in the dentate nucleus, putamen-caudate segment of the striate body, and the Purkinje cells of the cerebellar cortex. On the basis of these, and similar reports, they believe that it is reasonable to think of acute chorea as divisible into two groups, one characterized by more or less diffuse encephalitic lesions, and the other of a more degenerative type with vascular modifications.

The authors agree with Kinnier Wilson and others of the Hughlings Jackson school that destruction of a region of the central nervous system cannot account for positive phenomena such as spasms, contractures and choreo-athetotic movements; that all it can explain are phenomena of deficit. They accept, too, the idea of "diaschisis," in a general and dynamic way, whereby a lesion at one point may arouse inhibitions, or releases therefrom, in centers at a distance. But when it comes to agreement with the idea, seemingly experimentally substantiated, that chorea is due to direct pathologic change of the motor cortex, they object that the cessation of choreic movement in hemiplegia really proves nothing except that in hypertonic states such movements are impossible. They do concur, however, in the idea that it is impossible to conceive of choreic movements without the existence of a cortex. This is shown by the absence of anything simulating choreic movements in decerebrate rigidity; also by the generally accepted view

that such movements cease during sleep and hypnosis. An hypothesis is ventured that the muscular disorder is influenced by disturbance of the cerebellothalamic tract.

ANDERSON, Los Angeles.

THROMBOPHLEBITIS OF THE CAVERNOUS SINUS. G. WEILL, Rev. d'oto-neuro-opht. **7**:737 (Dec.) 1929.

Clinical and autopsy observations in four cases of cavernous sinus thrombosis, seen within two years, together with critical comments, comprise this report. The patients in the four cases were aged 23, 27, 32 and 35, respectively, were strong and robust, and yet died within a few days from septicemia. The source of infection in two cases was a common furuncle in the frontal region and on the neck; in the third it was a mastitis with metastasis in the tonsils, and in the fourth there was a maxillary sinusitis complicated by erysipelas. Examination of the pus and blood cultures in all four cases revealed staphylococci. In none of the cases was there a collection of pus; even in the orbit, in spite of a pronounced exophthalmos, no true abscess was found. On admission to the hospital, exophthalmos was present in all; in one case it was bilateral from the beginning, while in the others the second eye became involved only twenty-four hours before death.

The differential diagnosis between a simple phlegmon of the orbit and suppurative phlebitis of the orbital veins is always difficult, except in cases with bilateral exophthalmos. High fever and albuminuria are found in both diseases. The absence of a pus collection in the orbit is always an unfavorable sign. In these cases the exophthalmos is due to a pronounced engorgement of the veins of the orbit from the infectious phlebitis rather than to purulent infiltration of the cellular tissue. Neither does the presence of thrombosed subcutaneous veins always decide the question as between simple orbital phlegmon and sinus thrombosis since the former has been observed in a case of staphylococcemia arising from a furuncle of the nose. Cerebral symptoms in cavernous sinus thrombosis occur late, usually only a few hours before death.

The difficulty of an early differential diagnosis is a handicap in both treatment and prognosis. Surgical intervention is useless in cavernous sinus thrombophlebitis, owing to its inaccessibility. When doubt exists as to the presence of phlebitis and orbital abscess is suspected, wide incision in the border of the orbit (rather than punctures which are dangerous) is indicated early and it should be carried down to the periosteum with elevation of the latter. Expectant treatment until a visible abscess is present is condemned.

Orbital phlegmon is a dangerous malady, especially if complicated by erysipelas. It is most often due to a propagation of the infection from chronic sinusitis.

The development of the phlebitis in the first case is interesting. In a woman, aged 23, mastitis developed following a normal labor. This was widely opened, but two days later, high temperature, stiffness of the neck and purulent angina appeared. Twenty-four hours later, there were exophthalmos with chemosis in the left eye, albuminuria and prostration. Pus from the throat, from the abscess of the breast and blood culture showed, on examination, *Staphylococcus aureus*. Death occurred on the third day after admission.

DENNIS, Colorado Springs, Colo.

FURTHER RESULTS OF MALARIA TREATMENT IN DEMENTIA PARALYTICA. E. MEYER, Arch. f. Psychiat. **88**:234, 1929.

The author reviews experiences with the treatment for dementia paralytica with malaria in his clinic. From 1923 until 1928, 309 patients with dementia paralytica were treated. Two hundred and seventy of these patients were closely followed-up. Of these, 17.7 per cent showed a complete recovery which has persisted to date; 15.5 per cent showed a good improvement with return to work;

13.7 per cent showed some improvement, although retaining some defects; 22.9 per cent showed no improvement, and 30.3 per cent died either as a result of the treatment or without direct relation to it. In reviewing the literature, Meyer finds that these proportions correspond closely with those reported by Gerstmann in 400 cases and Schultze in 1,000 cases. He finds that the recent statements by some authors that recovery in this disease occurs spontaneously almost as often as when malarial therapy is employed, are not justified. Even the most optimistic reports in this direction show no more than about 20 per cent of improvements as compared with 47 per cent of his own. This is true also of the treatment for dementia paralytica by other methods.

At present it can be stated definitely that the malarial treatment is undoubtedly the best method of therapy in dementia paralytica. The changes in the blood and cerebrospinal fluid are not consistent, and at any rate they do not seem to run parallel with the clinical improvement. The author regrets that so far there is no reliable criterion of prognosis in this method. (Apparently he has not followed the condition of the blood cerebrospinal fluid barrier in this connection.—Abstracter.) The deaths were due mostly to the progress of the dementia paralytica, or to some intercurrent disease, although several were directly due to the malarial disease. The author finds that heart disease, increased blood pressure and involvement of the kidney do not necessarily contraindicate this treatment. When they are definitely established, however, great care must be exercised. Tuberculosis of an advanced type, a poor state of nutrition, involvement of the bladder, marked focal signs, such as paralysis, aphasia, etc., as well as marked excitements, must be considered as contraindications to malarial treatment. In some cases in which such contraindications exist, it may be advisable to allow only three or four paroxysms at a time and then to repeat the malarial treatment as many times as is deemed necessary.

In cases of tabes, the results were not as promising. Of twenty-one patients, only eight showed improvement, but there were no deaths. In cases of cerebrospinal syphilis the treatment is more effective. In fifteen patients, nine showed definite improvement. However, one must remember that other methods of treatment, such as the application of iodides, mercury, bismuth and arsphenamine, frequently bring about good results.

MALAMUD, Iowa City.

HEREDITARY SYPHILIS IN OTO-NEURO-OPHTHALMOLOGY. G. WEILL, Rev. d'oto-neuro-opht. **7**:669 (Nov.) 1929.

It is rare to find ocular manifestations of syphilis in the new-born, although syphilitic coryza is regularly found at this period. The ocular lesions develop later under two forms: 1. Chorioretinitis, which is discovered only by the ophthalmoscope and, owing to the difficulty of the examination, is often undiscovered until much later. It is a very important sign and permits a diagnosis of hereditary syphilis in otherwise doubtful cases. The period of its manifestation extends from the first few months to the age of 4 or 5 years. 2. Late congenital syphilis occurs in the period extending from the age of 5 or 6 years to beyond puberty, ocular manifestations occurring even at 30 years of age or more. It is characterized by interstitial or parenchymatous keratitis, and is of great interest to neurologists and otorhinologists as well as to internists. This condition is often accompanied by gummatous osteitis of the nasal septum, labyrinthine deafness and dacriocystitis.

The great interest attaching to interstitial keratitis and chorioretinitis is that both leave sequelae which persist during the life of the patient, the former as deep vascularization of the cornea, the latter as peripheral pigmentary deposits in the retina.

On account of its interest in the three specialties as well as in general medicine, attention is called to syphilis in the third generation. Some authorities believe in a true transmission of syphilis to the offspring of persons with hereditary syphilis, while others maintain that the syphilitic infection merely prepares the ground for all sorts of diatheses, dyscrasias, cachectic states and endocrine disturbances.

Attempts have been made to decide this question by researches, especially by oculists, on the ultimate fate of children of persons with proved cases of hereditary syphilis. Treacher Collins found, among twelve mothers afflicted with parenchymatous keratitis, that there were sixty living children and five miscarriages. Twenty-six of the children died in the first year of life; twenty-five of the remainder were well, and nine had various dystrophies. Igersheimer reported on fifty-seven children of parents with interstitial keratitis, of whom ten died at an early age, one was stillborn and there were eight miscarriages. Among twelve of the children, the Bordet-Wassermann reaction was positive only in one. Sidler Huguenin examined sixty-five children of parents with interstitial keratitis by roentgenograms of the epiphyses of the knee joint, and made Wassermann tests. The latter was found positive only once, and the films were all negative.

The author's investigations of a small number of patients revealed no manifest signs of syphilis but they were, in general, thin and sickly. His conclusion is that persons with hereditary syphilis produce children in whom the Wassermann reaction is negative but who present various pathologic changes of the endocrine system.

DENNIS, Colorado Springs, Colo.

CONTRIBUTIONS OF OTOLOGY TO NEUROLOGY. J. G. WILSON, Arch. Otolaryng. **11**:265 (March) 1930.

This excellent article must be read in toto to be appreciated properly. Wilson points out that the chief advances in otology have been along the lines of physics and biologic sciences. The various audiometers represent the former, and the vast amount of work on the labyrinth represents the latter. He mentions especially the work of Seashore, Magnus and Maxwell. The American Otological Society has played an important part in these researches by indicating the needs, and by its endowments. Wilson discusses especially the studies on the vestibulo-ocular reflex in intracranial lesions.

Vestibulo-Ocular Reflex.—In order to keep a definite posture, information of alterations of posture must be carried to the brain. These in part constitute the vestibular reflexes. Destruction of the labyrinth or stimulation of the ear with cold water gives the slow component of nystagmus to the affected side. "If the lesion is confined to the labyrinth, the essential point is the deviation of the eyes to the side of the lesion." Labyrinthine nystagmus is of three or four days' duration. Cerebellar nystagmus may persist for years. "In lesions of the peripheral organ of the ear, the spontaneous vestibular reflexes observed are to be regarded as due to a loss of function and not to an increase of function, supposedly owing to irritation. If such an increase of function occurs, it must be rare, as I have never observed it. It would appear as a deviation of the eyes to the side opposite the lesion." Lesions of the lateral lobe of the cerebellum cause deviation of the eyes to the side opposite the lesion, as is shown in a case quoted by the author.

This difference in direction of the slow phase in some cases with involvement of the labyrinth is not thoroughly understood. The author believes that it is due to a loss of cerebellar influence on the vestibular nuclei of the same side. He then describes the ataxic movement of the eyes in cerebellar nystagmus and cites a case. It is more marked on looking to the side of the lesion. A lesion in or near the mesencephalon, involving this area by direct or indirect pressure, is often marked by a vertical nystagmus. One gets dissociation of eye movements and perverted nystagmus in such lesions. In a case quoted, with vertical nystagmus, the lesion was found in the upper part of the brain stem. Another case was diagnosed as tumor of the midbrain, probably pineal in origin. He notes that in lesions above the brain stem nystagmus comes on after slight irrigation, lasts longer, with rapid oscillations, and has severe reactions. He notes the influence of the cerebrum on the quick component and quotes a case to illustrate this. He also quotes the results obtained by de Kleijn in rabbits in which one cerebral hemisphere was extirpated.

HUNTER, Philadelphia.

THE NONEXISTENCE OF IRRITATIVE DISTURBANCES IN CEREBRAL PATHOLOGY. D. TRIANTAPHYLLOS, Encéphale **25**:127 (Feb.) 1930.

The writer comments on the general acceptance given to the idea that epileptic symptoms are irritative phenomena, in spite of the contributions of Hughlings Jackson, to whom he makes due acknowledgment. He makes the blanket assertion that augmentation of function as a result of disease does not exist in the nervous system, either central or peripheral. Then he applies the principle specifically to epilepsy.

In discussing the sensory aura, an hallucinatory phenomenon, the general point of view has been that it represents excitation of certain sensory centers. Yet it is a fact that even in tumor (which is considered an irritative lesion, par excellence) of Broca's area, nothing is provoked but aphasia, which is certainly a phenomenon of deficit. Even if hallucinations exist in cases of tumor of the cortical visual centers, this is not proof of excitatory stimuli, but only that the neoplasm provokes mental disturbance of which hallucination is one manifestation. Furthermore, since hallucinations are most frequently observed in diseases with essentially destructive lesions, a logical paradox is encountered in trying to explain as irritative a phenomenon manifested most clearly in hypofunctional states. As to the motor aura, i. e., spasms and contractures, these are but momentary releases from the inhibition of the cortical centers. When the eyes, in conjugate deviation, "oppose" the lesion in the frontal lobe, this has been considered due to suspension of the inhibitory function of the cortical centers in question, permitting hyperfunction of the mesencephalic supranuclear center of the opposite side.

When the topic of painful sensations is encountered, greater difficulty of explanation is admitted. Here, too, analysis of the situation seems to warrant the view that the so-called hyperalgesia in neuritis is really due to cortical interpretation of neuritic pain, plus the stimulus pain arising from the adjacent intact fiber. That is, in such seeming hyperalgesia, at least two cortical cells are thrown into activity, as opposed to only one normally. It is generally admitted that it is impossible to augment fineness of touch or of deep sensibility; why should pain sensibility be different? Furthermore, coexisting with the painful hyperalgesia in neuritis is a diminution of motor and reflex function — which means essentially the coexistence of hyperfunction and hypofunction of the same area, and which presents at the best a problem difficult to elucidate by the traditional way. Psychic disorders, such as mania, have been inadequately explained as irritative. Both the alternations between manic and depressive states, and the long duration of certain "irritative" states, point to the improbability of this explanation.

The author concludes by hazarding a theory concerning the applicability of this conception to all types of body cells.

ANDERSON, Los Angeles.

THE PATHOGENESIS OF FRIEDREICH'S DISEASE. V. Rabinowitsch, Ztschr. f. d. ges. Neurol. u. Psychiat. **122**:462, 1929.

The pathogenesis of Friedreich's disease is not yet known. The prevailing views consider it as a constitutional disease caused by certain peculiarities in heredity. There exists, therefore, an injury of the neuro-epithelial germinal layer, which sooner or later leads to a gradual decay of certain parts of the nervous system. Certain authors oppose the abiotrophic theory and argue in favor of a toxic infectious origin of Friedreich's disease. In favor of this theory is the fact that intercurrent febrile diseases can cause an exacerbation of a Friedreich's ataxia. Stern has described cases which have developed after measles. Dawidenkow argued that a notoriously infectious disease, Westphal-Leyden acute ataxia, has many points in common with Friedreich's disease. Finally, it is known that Friedreich's ataxia may develop on the basis of congenital syphilis.

Anatomically, in Friedreich's disease there is injury of the neuro-epithelial portion of the nervous system, while the vessels escape injury, and inflammatory phenomena are absent. In other organs, however, there is injury of the deriva-

tives of the mesenchyme. The heart is often involved either in myocardial disease or in endocardial disease. The involvement of the heart is often a cause of death in Friedreich's disease. The skeletal muscles escape in typical cases, but muscle atrophies are often seen. Often the bony skeleton is diseased, with kyphoscoliosis and deformity of the foot. These bony changes are usually looked on as secondary, but Jendrassik considers them primary in the same sense as the changes in the nervous system. Often the bony changes are apparent before the nervous signs.

Rabinowitsch reports an atypical case of Friedreich's ataxia and on this basis draws some conclusions on the pathogenesis of this disease. His case was familial, and the patient presented locomotor ataxia, hypotonia of the lower extremities, a Babinski phenomenon, a deformity of the foot and the absence of sensory signs. The heart sounds were distant, and the sclerae had a blue color. The lungs were emphysematous, and the lower bundle of the right pectoralis major showed an aplasia.

Rabinowitsch interprets these signs to indicate a constitutional weakness of the derivatives of the mesoderm. This assumption makes understandable the blue sclerae, the symptoms of the heart and the lungs, the developmental inhibition of the skeletal muscles and the bony deformities. Its relation to the nervous system is explained by the fact that the mesodermal elements in the nervous system permit the action of toxins on the nerve elements and so cause nervous symptoms. There follows an intoxication of the nervous system through toxic metabolic products and hormones.

ALPERS, Philadelphia.

THE CLINICAL AND PHYSIOPATHOLOGIC SIGNIFICANCE OF THE SIGN OF CHVOSTEK. J. PAVEL, J. CLAUDIAN and D. CORNATEANO, Ann. de méd. **27**:86 (Jan.) 1930.

There is no uniformity among clinicians as to the technic of the Chvostek test. Usually the sign is called positive if there is a sudden contraction of the facial muscles of the same side after percussion of a point at the middle of a line between the labial angle and the external auditory meatus. Other investigators percussed the preauricular zone and obtained a contraction of the corresponding half of the face if the sign was positive. The authors call the latter phenomenon Chvostek I. By a positive Chvostek II they understand a contraction of the muscles which raise the upper lip and the lateral wall of the nose after percussion of the area below the zygomatic protuberance. Chvostek III is the isolated elevation of the upper lip after percussion of the same point. To the confusion in the practical application of the test is added the uncertainty as to its existence under normal conditions. In cases of tetany the sign cannot be obtained in children aged from 3 to 6 months. Instead, there have been described a number of pathologic reflexes at this age, which are supposed to indicate an acute or latent tetany: the signs of Escherich, Thiemich and Moro and the sucking reflex. After 6 months, up to the age of 3 or 4 years, a positive Chvostek sign is considered as indicative of tetany. After this age the sign loses its significance, and, if positive, points only to a "neuropathic constitution" which is connected with a certain vasomotor excitability (Feer).

In their own investigations the authors found the Chvostek signs I and II positive in different diseases at all ages from 3 to 30 years in from 10 to 27 per cent of cases which did not show any other clinical signs of tetany. In addition, the sign was found positive in a number of normal persons. In other cases it could be obtained only on one side of the face, and it was absent in some evident cases of tetany. Electric stimulation of the facial nerve in cases presenting a positive Chvostek sign did not indicate any relationship between the two phenomena. The classic opinion that a positive Chvostek sign in sucklings always points to a latent tetany has been challenged by Kleinschmidt and Roedel and by Heuyer and Longchamp, who did not see the positive sign in cases of typical tetany. Of late there has been the tendency for many neurologists and pediatricians to diag-

nose "spasmophilia" or a latent tetany if the Chvostek sign was positive in children or adults without any clinical signs of tetany. The authors oppose this tendency and summarize their report by denying that the sign of Chvostek has any significance in the diagnosis of tetany.

WEIL, Chicago.

CEREBRAL UREMIA. BLINDNESS. TRANSIENT PAPILLARY STASIS. UREMIC MENINGEAL REACTION. H. ROGER, AUBARET and ALBERT CRÉMIEUX, Rev. d'oto-neuro-opht. **7**:336 (May) 1929.

This case report concerns a man, aged 33, who, a few months previously, had attacks of indigestion, vomiting and asthenia. Shortly afterward he became dyspneic during effort and at night, the attacks often resembling asthma. An examination by another physician revealed: the urine contained 0.5 of albumin; the urea in the blood was 0.59; the Wassermann reaction was negative. The spinal fluid showed: 1.15 of albumin, leukocytes 0.9, Wassermann reaction negative, chlorides 6.43, sugar normal. The blood pressure (Vaquez) was 24. Following phlebotomy of 0.25 liter there was great excitability with inarticulate cries and lowered vision. Two weeks later, there was another crisis, with intense headache and terrifying hallucinations. There were almost total blindness and bilateral choked disks. Antisyphilitic treatment was given which seemed to improve the vision, but the cardiorenal trouble remained, and there were a large liver, dilated ventricles and hypertension, 25 systolic and 14 diastolic. The past history showed that the patient was married in 1918, but that no children resulted. He had suffered the loss of some toes from freezing during the World War. He was an absinth habitué.

On entrance to the hospital the patient was stupid, responded poorly to questions and did not cooperate in the examination. He was emaciated, pale and agitated. An examination of the eyes revealed complete blindness, bilateral midriasis and external strabismus; the pupillary reflexes were abolished. There was no sign of papillary stasis, but in the left eye there appeared a small spot of retinitis. The tendon reflexes were a little lively, without clonus. The Oppenheim test gave positive results. There was pain on eliciting Laségue's sign, which was limited to 45 degrees. There was a slight pericardial rub, and the blood urea was increased (1.90). The urine showed a slight trace of albumin but no sugar. The cerebrospinal fluid was under slight tension and was xanthochromic; the leukocytes were 7.4. The Bordet-Wassermann reaction was negative in the spinal fluid and blood. The patient grew worse; the evening temperature reached 39.9 C. (103.8 F.), and death occurred on the eleventh day after admission. There was no autopsy.

Most interesting were the transitory choked disks, which pointed to a tumor. It was probably due to a serous meningitis. Uremic patients often suffer from meningeal accidents of mechanical rather than toxic origin. In this case there were small meningeal hemorrhages and meningocerebral lesions. The oculist maintains that there was an albuminuric neuroretinitis counterfeiting a choked disk.

DENNIS, Colorado Springs, Colo.

A CASE OF TUBEROUS SCLEROSIS. PACHECO E SILVA and BONIFACIA DE CASTRO FILHO, Memorias do Hospital de Juquery, 1928-1929, vols. 5 and 6, nos. 5 and 6, p. 109.

In the Brazilian literature cases of tuberous sclerosis are rare. Most characteristic of the condition is the variety of organs affected. In 1880, the first observation of this malady was made by Bourneville and Brissaud, who gave it its present name. They regarded it as a sequel to an inflammatory process, and the term tuberopolioencephalitis clung to it till Pellizzi, Vogt, Geitlin and Bielschowsky regarded it as a peculiar form of neoplasm. Various psychic disturbances, such as dementia, idiocy and character anomalies, have been described. Because of the congenital lesions associated with it, it has come to be regarded as a dysgenic disorder. Harelip, malformation of the palate, omphalocele, extra

fingers, genital malformation and oxycephalus are some of the congenital lesions observed. In the skin, sebaceous adenomas are frequent. Those who uphold the dysgenic theory claim a disturbance of the development of the neuroblasts and spongioblasts before their migration to the cortex. Irregular development of the nerve cells and poverty of the nerve fibers in the cortex have been reported in other dysplastic conditions of the nervous system, but the appearance of giant cells and tumors of the ventricles is peculiar to this condition. Bielschowsky attributed the condition to a disturbance in the embryonic development of the glia, with failure of complete differentiation between the neuroblasts and the spongioblasts. The scleroses show no regularity, though there is a predilection for the frontal and temporal regions. They are usually the size of a pea, but can be large enough to cause death. Microscopically, they show an irregular network of glia fibers with a reduction of neuronic elements that is more marked in the central than in the peripheral portions of the nodule. Disproportionately large glia and nerve cells are usually found. Clinically, the picture is that of infantile encephalopathy, with chiefly intellectual deficiency and motor disturbance, usually in the form of epileptiform seizures. In the absence of other congenital anomalies and particularly sebaceous adenomas, the differential diagnosis from epilepsy is often impossible.

The case of a girl, aged 15, with severe mental defect, epileptiform attacks and sebaceous adenoma is described as showing at autopsy numerous tuberculous areas of soft consistency distributed irregularly over the brain, including the lateral ventricles. The kidney showed degenerated tubercles. Microscopically, the characteristic picture already described was found. The heart showed white plaques of fatty degeneration. The other organs were essentially normal.

HART, Greenwich, Conn.

THE FASCICULUS CENTROPARIETALIS. E. KATZENSTEIN, Schweiz. Arch. f. Neurol. u. Psychiat. **25**:3, 1929.

The existence of the fasciculus centroparietalis as a distinct bundle of fibers in the centrum semiovale, as described by von Monakow, is doubted by Niessl von Mayendorf. The author gives the results of developmental, experimental and pathologic studies on the question which were conducted in the former's laboratory. In the introduction he summarizes the views of Flechzig and Brodmann, among others, on fiber systems and centers for projection and association in the cerebrum, and gives a brief exposition of von Monakow's views in regard to the importance of the time factor in the development of cerebral function.

In studying serial sections of nine brains of infants and young children, an area was noted in the centrum semiovale between the corona radiata and the "U" fibers, extending from the posterior frontal to the parietal regions, which was paler than the surrounding areas and was made up of fine sagittal fibers that became myelinated relatively late. A similar area, because of its finer structure and relative pallor, could be discerned in two normal adult brains. In one of these, that of a gifted pianist, the area was relatively large; this fact, along with the late myelinization of the fibers in question, is believed by the author to indicate that this fasciculus subserves acts requiring considerable skill and training. The same bundle could be demonstrated by secondary degeneration in brains of experimental animals, including monkeys.

Serial sections stained with carmine and also by the Pal method from twelve pathologic cases were studied. In all of these either the precentral and postcentral gyri or the parietal areas had been injured, either through vascular accidents or trauma. Clinically, the cases presented hemiplegias, aphasias, apraxias and ataxias with a marked tendency to regression of symptoms. In all instances the existence of a fiber system extending principally from the precentral and postcentral convolutions to the angular and supramarginal gyri could be demonstrated either by the presence of secondary degeneration in the earlier cases or by atrophy of the corresponding area when a number of years had elapsed since the injury. In

general, there was considerable glial proliferation in the brains examined from one to three years after the accident and changes of a more regressive character in those seen later.

From his studies the author concludes that the existence of the fasciculus centroparietalis is justified. The regression of symptoms generally noted argues, he believes, for von Monakow's theory of diaschisis.

DANIELS, Rochester, Minn.

GERONTOPHILIA. A. KUTZINSKI, Monatschr. f. Psychiat. u. Neurol. **74**:86 (Nov.) 1929.

The author regards gerontophilia as a form of assertion of the infantile need for support from an older person, as indicated by the compulsive choice of a very much older homosexual or heterosexual love object. He reports the case of a middle aged man whose father died when the patient was 3 years of age, and whose mother soon married again into a different religious faith. When about 10 years of age, after receiving some sexual information from older boys, he made sexual advances toward his mother whom he accidently saw exposed. For this he was beaten by his stepfather with whom he never got along well. As a result of this experience, strong feelings of guilt developed, and not long afterward he broke off all relations with his immediate family, ostensibly on the grounds of religious differences. Masturbation occurred almost daily from the age of 11 to 19 and was terminated with the onset of heterosexual relations. For the latter the patient picked out women with sharp features similar to his mother. There was one isolated homosexual episode, apparently of a minor nature, at the age of 18. His dreams frequently concerned his mother, often in an outspoken erotic manner. At the age of 24 he married, and he had six children; he served in the army during the war. Following complete sexual abstinence for over a year, he encountered, while bathing, an elderly woman with whom he had sexual relations. He lost all love for his wife, showing instead outspoken erotic impulses toward elderly women, which were so compulsive in nature that they rendered work impossible.

The author believes that his case illustrates very well the mechanisms postulated by Freud for this condition. The sexual trauma occurring when erotic impulses were awakening resulted in a mother fixation which remained repressed, and later, under the pressure of prolonged abstinence, became transformed into erotic impulses toward elderly women. He agrees with Stekel, however, that the basis of gerontophilia may vary in different cases, for in addition to the foregoing mechanism it is sometimes traceable to feelings of inferiority; in other instances it represents a disguised form of homosexuality. It was noteworthy that the patient's individual experiences were all perfectly conscious, but not their relationship. After pointing out the connection between the gerontophilia and the early mother fixation, there was a rapid improvement. Therapeutic success seemed complete, but the author does not think that sufficient time has elapsed to pronounce the cure permanent.

ROTHSCHILD, Foxborough, Mass.

HETEROPLASTIC TRANSPLANTATION OF EYES BETWEEN THE LARVAE OF TWO SPECIES OF AMBLYSTOMA. L. S. STONE, J. Exper. Zool. **55**:193 (Jan. 13) 1930.

Return of circulation in the transplanted eyes was observed as early as the first day after operation, and in no case later than the sixth day. Owing to the loss of blood cells and fluids at the time of operation, the transplanted eyes underwent a temporary decrease in size. An increase occurred as soon as circulation was reestablished. Larval eyes grafted from one species to the other followed closely the growth curves of their donor controls. Under ordinary conditions of feeding, tigrinum eyes on punctatum larvae grew more rapidly than the normal eyes of the host, and, up to the time of the metamorphosis of the hosts, they

exceeded in size the donor control eyes. Punctatum eyes on tigrinum hosts during the same period were smaller in size than their normal donor controls. The size of the lens of the punctatum transplant was proportional to the size of the eye for a long period. In the transplanted tigrinum eye, the lens size was proportional to the eye size up to the time of metamorphosis of the host. Later, the lens size remained greater in proportion to eye size. The changes in the pigmentation of the iris from the larval to the adult condition in the transplanted eyes in both species depended, as in their normal controls, on the metamorphosis of the hosts. The pupil of the normal larval punctatum eye showed no measurable contraction and expansion to light and darkness, and that of the normal larval tigrinum eye showed only slight reactions during late larval life. Metamorphosis of the host brought about marked pupillary reactions in the transplanted and normal eyes in both species. The reactions were hastened in eyes on punctatum hosts. There was a gradual increase in the thickness of the cornea and retina in the normal and transplanted eyes of both species as the eyes increased in size. The number of cells in the ganglionic layer was less per unit area in the transplanted eyes than in their respective normal donor controls due to varying amounts of ganglion cells disintegrating during the first two weeks after operation. Susbsequent to the first two weeks after operation, it was certain in only two transplants (one in each species) that no optic nerve connected the eye with the brain. This had no effect on the growth of these eyes. Injured muscle fibers appeared to block the course of growth of the regenerating optic nerve in both cases. Return of vision was proved in six cases prepared for testing their function. These eyes showed corneal and pupillary reflexes, ocular movements and pigmentation changes as described for other transplants and normal controls.

WYMAN, Boston.

INHERITED NEUROSYPHILIS. MABEL G. MASTEN, J. Nerv. & Ment. Dis. **70**:379 (Oct.) 1929.

Little of moment has been added to the knowledge of inherited syphilis since the days of Hutchinson and Fournier. In 1927, Morton Smith reviewed the cases of 1,000 patients treated in the Massachusetts General Hospital, but no conclusions were drawn as to the evidence of neural syphilis. Statistics vary according to the source of material and completeness of examination. The percentage of involvement of the nervous system in congenital syphilis has varied from 10 to 43. It is probable that the incidence is much higher than commonly held because the signs are few and spinal fluid examinations in children are generally neglected. As in adults, the Wassermann reactions of the blood may be negative in late congenital syphilis while the spinal fluid may be positive. The question of a neurotropic virus has always been debatable and the involvement of the nerve tissue is probably the result of a number of factors, some of which are at present unknown. The most common form of inherited neurosyphilis is paralytic dementia. Ten of the twenty cases reported by the author were paralytic dementia, among which two showed optic atrophy. Juvenile paralytic dementia does not show the distinguishing types of the acquired form. The usual picture is that of simple deterioration and the child may later become restless, muttering and repulsive. Delusions, when present, are simple, silly and transient. Congenital tabes is much less common than paralytic dementia, but five of the series reported were tabetic with gastric crises and optic atrophy but less marked ataxia. Six cases showed definite basal systolic murmurs; three of them had syphilitic involvement of the aortic arch. The pupils were fixed and dilated in nine of the series. When possible, serologic studies of patients should be made and the physician must remember that a Wassermann reaction, negative at birth, may be positive later in latent cases. Lumbar punctures, so often neglected in children, should be carried out in all suspicious obscure neurologic cases, but a negative Wassermann reaction of the spinal fluid does not exclude congenital syphilis; one of the most outstanding cases of tabes dorsalis in the series had a completely negative serology.

Treatment is considered as in no essential way different from that in adult cases; mercury, bismuth, sulpharsphenamine, neoarsphenamine and tryparsamide are all used, the last named being the favorite. Marked mental improvement in one agitated, demented girl followed eight injections of tryparsamide. Another patient with juvenile paralytic dementia had ten chills of malaria, and definite mental and physical improvement was reported. The use of mercury and tryparsamide after the malaria is recommended.

HART, Greenwich, Conn.

BULBAR LOCALIZATION IN RESPIRATORY DISTURBANCES. TOKIHARU HAZAMA, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **31**:103, 1929.

CASE 1.—A man, aged 36, became ill in March, 1925, with a sore throat, which was followed by weakness and paresthesias in the lower limbs. This progressed so that by August he was unable to walk without assistance. In the middle of October, there appeared difficult breathing and inability to swallow, bradycardia, flaccid paralysis and vasomotor disturbances of the distal ends of the extremities, generalized atrophy without fibrillary twitchings, nerve tenderness, disturbances of deep sensibility in the fingers and toes, absent deep and superficial reflexes, no electric responses in the nerves and muscles and involvement of the diaphragm. The palate moved freely. The diagnosis rested between polyneuritis and encephalitis. The patient died from pneumonia nine months after the onset of the illness.

Examination of the medulla revealed: A beginning inflammatory process in which the greatest involvement was found in the vessels adjacent to the dorsal vagus nucleus. In addition to the vascular dilatation and small hemorrhages, there was a degeneration of the ganglion cells in the middle and oral portions of the dorsal group of cells in the dorsal nucleus of the vagus. The nucleus ambiguus also showed severe cell degeneration but no evidences of an inflammatory process.

CASE 2.—A case of syringobulbia, of more than twenty years' duration, showed histologically, among other changes, a cleft, on one side of the medulla which had entirely destroyed the ventral two thirds of the dorsal nucleus of the vagus on that side. In this case there is no record in the history of any disturbances of respiration or of a slow pulse. The only bulbar symptom mentioned is slow speech, for which no anatomic basis could be found on histologic examination.

CASE 3.—A woman, aged 55, had a hypokinetic-hypertonic syndrome following encephalitis, which lasted nearly three years. One of the significant symptoms was a disturbance in breathing characterized partly by generally poor breathing and partly by inspiratory stridor. The anatomic substratum for the respiratory changes was found in the dorsal portion of the dorsal nucleus of the vagus. The lesion consisted of a degeneration of the ganglion cells with some destruction of cells in that area and a slight edema. The locus cœruleus also showed a destruction of ganglion cells with no clue as to how this occurred.

From a study of these three cases and a review of the literature the author concludes that the dorsal portion of the dorsal nucleus of the vagus must be looked on as a center for respiration.

KESCHNER, New York.

CLINICO-ANATOMIC CONTRIBUTIONS TO THE PROBLEM OF THE CENTRAL REGULATION OF SLEEP RHYTHM. BELA HECHST, Arch. f. Psychiat. **87**:505, 1929.

The author reports four cases in which definitely localized lesions of the brain were associated with a disturbance of the sleep rhythm. Case 1 was that of a man, aged 27, in whom the disease started about three months before admission and whose outstanding symptoms were pronounced drowsiness, disturbance of eye movement and diabetes insipidus. Postmortem examination showed the presence of an ependymoma which affected the posterior third of the gray substance of the third ventricle, the gray substance of the sylvian aqueduct and the posterior and medial parts of the thalamus. Case 2 was that of a woman, aged 37, whose sickness began two months before admission and who also showed a marked

tendency to sleepiness, especially in the daytime. Autopsy showed a tumor (astrocytoma) which affected the posterior third of the left thalamus but very little of the central gray matter. Case 3 was that of a man, aged 42, whose illness began four weeks before admission and was also characterized by marked drowsiness and yawning. Postmortem examination showed a tumor affecting the medial part of the posterior third of the thalamus and the dorsal part of the gray matter of the aqueduct of Sylvius. In case 4 the patient had shown a tendency to drowsiness for six months before admission, and as this was the most outstanding feature of the clinical picture the diagnosis of a tumor at the floor of the third ventricle was made. At postmortem examination a tumor was discovered at the base of the brain between the optic chiasma and reaching into the third ventricle.

The author discusses the literature concerned with this problem and comes to the following conclusions: (1) The regulation of the normal sleep rhythm is carried on by the medial part of the posterior third of the thalamus, by the posterior third of the gray substance of the third ventricle and by the gray substance surrounding the aqueduct. These play equal rôles in this function. (2) The anatomic intactness of these centers is absolutely essential for normal sleep rhythm. (3) A disturbance of any one of the three centers will cause a pathologic drowsiness with a reversed rhythm. (4) One-sided lesions in these structures may also cause sleep disturbances. (5) The red nucleus, mammillary bodies and the corpora luysii are not associated with the regulation of sleep. (6) Destruction of the posterior part of the thalamus leads to disturbances of the emptying of the urinary bladder. (7) Diabetes insipidus is caused by an irritation of the tuber cinereum.

MALAMUD, Iowa City.

THE RELATIONSHIP BETWEEN TREATMENT OF SYPHILIS AND THE SUBSEQUENT INVOLVEMENT OF THE CENTRAL NERVOUS SYSTEM. S. B. GOLDBERG, Rev. Psychiat., Neurol. & Reflexol. (Leningrad) **4**:242, 1929.

The literature for the past few years devotes a great deal of attention to the treatment for syphilis and the appearance of metasyphilitic manifestations. In fact, the whole subject is so important that in 1928 it became the subject of discussion of the German Dermatological Association. No definite conclusions were made, but treatment was advocated on the basis of eradicating the dangers of the spread of the disease.

Two hundred and two patients exposed to syphilis were studied. The length of the incubation period between the primary sore and the appearance of the involvement of the central nervous system was used as a criterion. In the cases in group 1 no treatment was given. In group 2, the patients were treated with mercury, and the patients in group 3 received both mercury and arsphenamine. In addition, the patients in groups 2 and 3 were given potassium iodide.

In all of the patients the average incubation period between the infection and the onset of symptoms of involvement of the central nervous system was twelve years; however, in the patients in group 3, the average period was only seven and one-half years. In other words, the patients who were treated with both mercury and arsphenamine showed a shorter incubation period than those who were treated only with mercury.

The results seem to correspond with the experience of German authors, although Nonne pointed out that the danger of treatment with arsphenamine was due not to arsphenamine but to insufficient therapy, both in frequency and in total amount.

The author comes to the conclusion that even the most intensive treatment does not guarantee against the possibility of future involvement of the central nervous system. The patients showing syphilis of the central nervous system developed the symptoms most frequently after treatment with mercury and arsphenamine. The serologic picture does not always correspond to the clinical manifestations. The treatment for syphilis is always justified, if not on clinical then on social grounds.

KASANIN, Boston.

THERAPY OF PYLOROSPASM. EMILIE SCHROEDER, Fortschr. d. Med. **48**:93, 135, 177, 1930.

Physical therapy for pylorospasm includes the following: warm lead fomentations, diathermy and high frequency electrical treatment, all of which have their fervent advocates. Surgical therapy includes jejunostomy, resection of the pylorus, gastro-enterostomy and stretching of the pylorus. In 1910, Weber described partial pyloroplasty in which a longitudinal section through the serous and muscular coats is made and united by cross suture; the name given to the operation was submucous pyloroplasty. Under Rammstedt it became very popular. This was followed by a considerable division of opinion as to when operative measures should be adopted. A pyloric tumor is rather infrequently palpable in these cases, and Konrad and Kielmann described cases of congenital pyloric hypertrophy without clinical symptoms. Modern pediatrics finds no clear distinction between purely spastic and hypertrophic stenosis of the pylorus, and the consensus is in favor of operating only in the severer cases. Strauss, Leall and Heile recommend surgical intervention in cases in which from 70 to 80 per cent of the bismuth ingested remains in the stomach after four hours, but roentgenograms do not always give the correct estimation of the seriousness of the condition. According to Bloch, a rapid decline of weight is a vital indication for operation. To allow the condition to persist for a long time is apt to lower the child's resistance to various infections in the opinion of Finklestein. Hildebrandt and Strauss have preferred other operative measures as improvements on the Rammstedt operation. The great danger after the operation is vomiting and perforation peritonitis. Kirschner uses chloroform narcosis. Others use ether and even local anesthesia. Eckstein and Romminger used methylpropylcarbinolethyl cerbonate with the best results.

Ibrahim, in 1913, compiled the mortality rate from purely corrective treatment as 10.2 per cent in 400 cases, as contrasted with 10.6 per cent of 597 cases from operative methods.

HART, Greenwich, Conn.

THE RELATION OF THE DEVELOPING AUDITORY VESICLE TO THE FORMATION OF THE CARTILAGE CAPSULE IN AMBLYSTOMA PUNCTATUM. HELEN WARTON KAAN, J. Exper. Zool. **55**:263 (Jan. 13) 1930.

Cartilage may develop around an otic vesicle of *Amblystoma punctatum* when transplanted into the region directly anterior to the normal ear. This cartilage usually arises as an outgrowth from the trabecula or capsule of the host and may or may not form a complete capsule about the transplanted labyrinth. If transplantation takes place at a stage preceding invagination of the otic cup, the resulting labyrinth forms a much enlarged simple vesicle. At a later stage the resulting labyrinth shows increasing differentiation approaching that of the normal. A partial capsule may form also around the ear rudiment when transplanted to a position directly posterior to the normal ear. Transplantation of the otic vesicle into the gill region, anterior or posterior to the pronephros, to the limb region or the flank results in the formation of irregular vesicles without any trace of accompanying cartilage.

In all cases in which the otic cup or vesicle of *Rana sylvatica* was transplanted into *Amblystoma*, more or less cartilage of the *Rana* type developed near the transplanted vesicle. There was evidence of extra cartilage formation only in the region immediately surrounding the ear, from the trabecula and capsule of the host. Where transplanted *Rana* vesicles developed near other skeletal elements there was fusion between the *Rana* and the *Amblystoma* cartilage. This did not affect the development of the host skeleton. It is concluded that the developing ear can stimulate mesenchyme to form a cartilage capsule only in the region immediately surrounding the normal ear. A capsule forming about transplants into any other part of the body develops from cells introduced with the graft. Differences in development between the two species is suggested as the explanation

of the fact that mesenchyme cells were transplanted with the *Rana* vesicles, and not with those of *Amblystoma*. The effect of external factors, such as the presence or absence of mesenchyme, may explain the difference in development between ears in a normal and in an abnormal location.

WYMAN, Boston.

PARALYSIS OF THE DILATORS OF THE GLOTTIS: GERHARDT'S SYNDROME. V. FOTIADE, Rev. d'oto-neuro-opht. **7**:263 (April) 1929.

Gerhardt's syndrome is characterized by three cardinal symptoms: continual dyspnea during inspiration, accompanied by stridor; intermittent attacks of suffocation; intact voice. Laryngoscopically, the cords are in the paramedian position, they adduct on phonation but during inspiration they do not abduct beyond the paramedian position. There is pure paralysis of the dilators. Since Gerhardt's description of this condition, numerous observers have seen cases of abductor paralysis without the stormy clinical symptoms, and experimental paralysis of the dilators has been produced in animals unaccompanied by dyspnea. Four cases are reported, two of which were pure paralysis of the dilators. All patients had syphilis.

There are then two syndromes of abductor paralysis: (1) pure paralysis, which is well borne by the patient and is discovered only with the laryngoscope; (2) Gerhardt's syndrome, which includes the alarming symptoms of dyspnea. The anatomic substratum is not merely a paralysis of the dilators but is complicated by an irritation of the constrictor group of muscles, causing the cords to lie in the paramedian position with a glottis chink of only from 1 to 2 mm., which is insufficient for respiration. These are not two distinct morbid entities. Cases of pure dilator paralysis are met with in which the patient remains comfortable for a long time and then, at some given moment, Gerhardt's syndrome intervenes. The cause of this complication is not definitely known. The theory is advanced that there is a degenerative lesion in the nerve pathways and nuclei of the dilators in the bulb which causes an irritation in the adjacent centers of the constrictors. On the other hand, the work of Bertrand and Truffert shows the nonexistence of dilator and constrictor centers in the bulb.

Energetic and prolonged antisyphilitic treatment is advised, but one must bear in mind that in a case of simple latent paralysis of the dilators, a too energetic therapy can bring on Gerhardt's syndrome, probably a Jarisch-Herxheimer phenomenon.

DENNIS, Colorado Springs, Colo.

EPILEPTIC ATTACKS IN MULTIPLE SCLEROSIS. S. BAU-PRUSSAK and L. PRUSSAK, Ztschr. f. d. ges. Neurol. u. Psychiat. **122**:510, 1929.

Epileptic attacks are one of the rare complications of multiple sclerosis. Wilson and MacBride reported eight cases with such attacks. The question is important because of the diagnosis between multiple sclerosis and tumor of the brain. Not infrequently, operation is performed only to find later that the condition is one of multiple sclerosis. Jacksonian attacks, generalized epileptic convulsions, petit mal, epileptoid vertigo attacks with somnolence and epilepsia partialis continua have been observed in multiple sclerosis.

In 268 cases of multiple sclerosis observed since 1918, epileptic attacks occurred in only thirteen. In nine of these, the attacks occurred as an initial symptom, and in the other four they developed during the course of the disease. The time between the first epileptic attack and the development of other symptoms may be several years. In one case the epilepsy preceded other symptoms by twenty-five years, and in another case by about fifteen years. In a case of Wilson and MacBride, symptoms of multiple sclerosis developed eighteen years after the first epileptic attack. This is not a fortuitous occurrence. It may be thought that the epilepsy and multiple sclerosis are accidentally related, but that this is probably not so is indicated by the fact that the retrobulbar neuritis in multiple

sclerosis may precede other symptoms by many years. The diffuse gliosis which precedes the formation of plaques (Redlich) may be the anatomic substratum of the epileptic convulsions. The frequency of epileptic attacks varies greatly; in some cases they are rare, and in others frequent. In one case of Wilson and MacBride there were from six to seven attacks during an illness of twenty years. In the cases reported by Prussak there were jacksonian attacks, generalized convulsions, petit-mal and psychic equivalents. The cause of the epileptic attacks in multiple sclerosis is not clear. In any event, they constitute a part of the clinical picture and are not a coincidence as Oppenheim believed.

ALPERS, Philadelphia.

SOME CONTRIBUTIONS TO THE HISTOPATHOLOGY OF THE BRAIN. M. SHIMODA, Monatschr. f. Psychiat. u. Neurol. **74**:65 (Nov.) 1929.

In a rather brief and dogmatic communication the author gives his views on three constantly recurring questions in neuropathology. First, he discusses the so-called sclerosis of Ammon's horn in epilepsy which he found in only 13 per cent of his cases. He regards this sclerosis as a type of pseudomicrogyria produced by some process of disease generally occurring in childhood; as a rule it is inflammatory in nature, as evidenced by old inflammatory scars which he was always able to find in these cases. The fresh ischemic changes described by Spielmeyer have no relation, according to the author, to Ammon's horn sclerosis, which he thinks could not be caused merely by such temporary vascular spasm as might occur during a seizure. He warns against confusing the term Ammon's horn sclerosis with the much commoner Ammon's horn gliosis which he looks on as the anatomic basis of the epileptic character changes. It occurs as a reaction to the congestion produced in the brain during the convulsions.

In a few short paragraphs he takes the stand that the acute cell disease of Nissl is not the result of toxic or autotoxic processes. Furthermore, it does not bear any fundamental relation to catatonia. He believes that it is merely an expression of the acute or subacute exhaustion of the ganglion cells which may follow any condition of hyperactivity, sleeplessness and defective nutrition. In proof of this he cites a case of severe mania in which the patient died of exhaustion and showed the typical acute cell changes described by Nissl.

The problem of the origin of corpora amylacea is regarded by the author as solved by his work in 1918 and confirmed by later workers. In his opinion, corpora amylacea in the nervous system are formed by a circumscribed swelling of neurologia fibers. This is part of a peculiar involutional process in the glia which is dependent on the age of the patient rather than on any particular disease.

ROTHSCHILD, Foxborough, Mass.

INTRACRANIAL ANEURYSMS: THEIR RÔLE IN THE PRODUCTION OF OCULAR PALSIES. A. O. PFINGST and R. G. SPURLING, Arch. Ophth. **2**:391 (Oct.) 1929.

Intracranial aneurysms, the authors believe, are more frequent than is generally thought. They quote Cushing who says that a basal aneurysm is one of the common causes of sudden oculomotor palsy. The symptoms are those of pressure, localizing symptoms according to site and those peculiar to the aneurysm. The latter symptoms occur as a result of leak or rupture, and include dizziness, headache, pain and rigidity in the neck, and bloody spinal fluid. The important differentiations are those of aneurysm from syphilis, tumor and meningitis. The spinal fluid is bloody in the case of an aneurysm and shows a positive Wassermann test in syphilis; headache, pain and stiff neck are contrasuggestive of syphilis. Meningitis is recognized by its constitutional signs and special spinal fluid tests. Tumors are of slower development, and show more choking of the disks and less headache and stiff neck. The paper closes with two cases—each with repeated attacks of

headache, palsy of the third nerve and stiff neck; one with a bloody spinal fluid, and both showing gradual, incomplete recovery. The diagnosis was not verified by autopsy or operation in either case.

DAVIDSON, Philadelphia.

DIABETES INSIPIDUS AND LESIONS OF THE MID BRAIN: REPORT OF A CASE DUE TO A METASTATIC TUMOR OF THE HYPOTHALAMUS. T. B. FUTCHER, Am. J. M. Sc. **178**:837 (Dec.) 1929.

Views concerning the etiology of diabetes insipidus are reviewed in this article. In 1906, Schäfer, Magnus and Hering showed that the extract of the posterior pituitary lobe influenced water metabolism. Since the work of Cushing, in 1910, in removing the posterior lobe of the pituitary gland in dogs, the prevalent view has been that diabetes insipidus is due to disturbed function of the posterior lobe of the pituitary. The view that the polyurias associated with organic brain lesions were dependent on tumors involving the hypothalamus was first suggested by Erdheim in 1904.

The case of a man, aged 43, with diabetes insipidus, who had a primary carcinoma of the lung with metastasis to the hypothalamus of the brain without involvement of the pituitary, is reported. Two theories are maintained as to the formation of diabetes insipidus: (1) a mechanism through the nervous system, and (2) the deficiency of a specific diuretic-antidiuretic hormone. The fact that the posterior lobe of the pituitary and the structures in the hypothalamus are linked up by nerve fibers further complicates the etiology of diabetes insipidus.

MICHAELS, Detroit.

GANGLION CELL INCLUSIONS AND NUCLEAR DEGENERATIONS IN EPIDEMIC ENCEPHALITIS. R. NISHII, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **31**:96 1929.

Six cases of acute and chronic epidemic encephalitis were carefully examined by Nishii for ganglion cell inclusions. They all showed: (1) changes in the cell nucleus. These consisted of undoubted evidences of a severe involvement of the chromatic substance in the nucleus which had lost its netlike character and appeared in the form of a fibrous or granular structure. Some of the nuclei appeared as if they contained "inclusions" within them, but there was no doubt that one was dealing merely with a severe degenerative process in the nucleus. These chromatin masses took basic as well as acid stains. Simultaneously with this change, though independently of it, there was also noted (2) a change in the plasma, the chief characteristic of which was a delicate honey-comb-like appearance of the cell body, within which there were observed fine granules which stained well with toluidine blue as well as with eosin, although they could also be made out distinctly in iron-hematoxylin preparations.

Nishii has no doubt that these granular bodies within the cell are identical with the "minute bodies" described by Da Fano, and he believes that they are evidences of a degenerative process which runs parallel with the degenerative process in the nucleus.

KESCHNER, New York.

PARATHYROID THERAPY IN SCHIZOPHRENIA. KARL M. BOWMAN, J. Nerv. & Ment. Dis. **70**:353 (Oct.) 1929.

Disorders of the gonads, thyroid and other glands of internal secretion have been mentioned as possible etiologic factors in schizophrenia, but little attention has been paid to disorders of the parathyroid. Looney, in a study of eleven cases of catatonia, reported a slight decrease in the calcium content of the blood, as well as an increase in the calcium and decreased muscular rigidity following injection of parahormone.

The author reports three cases of catatonia in which were given injections of parahormone at first subcutaneously, later intravenously. Although the author

realizes that the number of cases is insufficient to draw definite conclusions, he reports his inability to secure any marked rise in the calcium content of the blood or any decrease in muscular tension. One case showed a sudden, spectacular improvement, not uncommon in catatonic states. Dr. Aubs' examinations of the blood at the Massachusetts General Hospital confirmed the laboratory work of the author. The author can see no clear relationship between the calcium content of the blood and and the parathyroid activity, on the one hand, and the catatonic state, on the other, but realizes the necessity for more work on the problem before it can be settled.

HART, Greenwich, Conn.

SPEECH DISORDERS. SMILEY BLANTON, Ment. Hyg. **13**:740 (Oct.) 1929.

After explaining the reason he believes speech to have a fundamentally emotional basis, Blanton lists the common types of speech disorders in children as delayed speech, oral inactivity, letter-sound substitution and stuttering. Delayed speech, he suggests, may be due to lack of physical or mental development, to illness, especially deafness, and to the lack of necessity for speech. By this he means that the child whose wants are satisfied before he makes verbal demands delays learning how to talk because he does not need speech. Blanton also mentions "negativism or hysterical conditions" as a cause for this sort of delay, but he does not define or discuss this factor. Oral inactivity, i. e., slurring and indistinctness of speech, he believes, always has some physical basis such as a poorly developed tongue, and very often rickets. By letter-sound substitution he means such phenomena as lisping. This, he suggests, represents a clinging to the infantile stage, and can be managed only by the psychologic maturation of the patient. Finally, he considers stuttering as the commonest and most important of the speech disorders. It is due to emotional maladjustments, says Blanton, and is not the cause of such maladjustments. Treatment consists in physical and mental hygiene, in emotional reeducation, in muscular coordination and in speech training.

DAVIDSON, Philadelphia.

SPHENOIDITIS — VALUE OF CYTOLOGIC STUDIES IN DIAGNOSIS. RULON S. TILLOTSON, M.D., Arch. Otolaryng. **10**:262 (Sept.) 1929.

In addition to roentgen examinations alone or with iodized oil, a cytologic study of the secretion is a valuable method of proving the presence of disease in the sphenoid. The oil is eliminated by the ciliated epithelia in from twenty-four to forty-eight hours. The rate of elimination may be an index of the health of the mucosa, although one must consider the position and the size of the ostium. If the normal ostium cannot be found, the author advises a trocar. It may be necessary to remove the obstruction by operation to gain access to the sinuses.

He reports twenty-five cases studied by introducing from 3 to 5 cc. of sterile warm water through the sphenoidal cannula into the sinus until it is well filled. The head is then thrown forward and the contents are aspirated. The aspirated fluid is centrifugated for five minutes. The author concludes that the normal healthy sinus contains no mononuclear or polymorphonuclear leukocytes; chronic infections show a preponderance of mononuclear leukocytes, and acute infections show a preponderance of polymorphonuclear cells.

The author quotes Sewall and Ersner in a similar study of maxillary sinus infection.

HUNTER, Philadelphia.

THE CEREBRAL CIRCULATION: IX. THE RELATION OF THE CERVICAL SYMPATHETIC NERVES TO CEREBRAL BLOOD SUPPLY. STANLEY COBB, Am. J. M. Sc. **178**:528 (Oct.) 1929.

The author traces the history of the study of vasomotor control from the indirect method of experiment to the more direct one. Hill, in 1896, concluded that there was no evidence for the causation of cerebral anemia by spasm of the

cerebral arteries. Weber, in 1908, experimenting with cats, found in 75 per cent a decrease in brain volume following stimulation of the cervical sympathetic nerve. In a series of experiments on cats, Talbott, Wolff and Cobb (1928) cut the cervical sympathetic nerve on one side, and injected berlin blue into the arterial system. Sections of the hemispheres were hardened, embedded and cut, and micrometer measurements were made. They found an increase in the capillary bed of the ipsilateral cerebral hemisphere.

Forbes and Wolff, in 1928, using a window in the skull as a means of experimentation, made direct observations on the contractions of the pial vessels. All this leads to the conclusion that the blood vessels of the brain react in the same way as vessels in other organs. Cobb concludes that, at present, vascular spasm has a much more reasonable working hypothesis than thirty years ago.

MICHAELS, Detroit.

A CASE OF RELAPSING INTERSTITIAL POLYNEURITIS. WILFRED HARRIS and WILFRED D. NEWCOMB, Brain **52**:108 (April) 1929.

A case of polyneuritis hypertrophica is presented which differs from all those previously reported in that the chief clinical feature was recurrent attacks of polyneuritis over a period of six years, with complete recovery between the attacks of paralysis. The last attack, however, was progressive during twelve months, and at the end, diaphragmatic paralysis occurred. The authors assume that the Dejerine and Sottas type of familial hypertrophic interstitial neuritis should be distinguished as a separate group from recurrent polyneuritis, to which their case belongs. The interstitial neuritis found in the nerve trunks and spinal nerve roots in their case, it seems, forms a link between the two types. It is conceivable that a similar cause may be responsible for both conditions. The likely factor is an autotoxemia from an enterotoxin, analogous to that found in cases of hemotoporphyrinuric polyneuritis.

BECK, Buffalo, N. Y.

CONSTITUTIONAL VARIATION AND SUSCEPTIBILITY TO DISEASE. WADE H. BROWN, Arch. Int. Med. **44**:625 (Nov.) 1929.

Disease is an interplay of two variables, the etiologic factor on one hand, and the patient's constitution on the other. Brown concerns himself with variations in constitution and endeavors to apply a quantitative scientific technic to the study of these variations. His thesis is that function precedes and determines structure, so that the organ relationships are ordinarily at an optimum. When submitted to environment, however, changes, in both degree and kind, occur in these organ relationships. For the study of this, Brown worked on more than 600 rabbits. He discovered that the greatest variations occurred in lymphoid tissues and the next highest degree of inconstancy was found in the endocrines. He then studied these relationships in disease. He also emphasizes the seasonal variations in organ relationships. His paper is clarified by numerous charts and tables.

DAVIDSON, Philadelphia.

ARTERIOSCLEROSIS AND MANIC-DEPRESSIVE PSYCHOSES. T. SONDÉN, Acta psychiat. et neurol. **4**:217, 1929.

Arteriosclerosis develops exceptionally before the age of 30, and even between 30 and 39 it is rare. From the age of 40, its incidence increases rapidly and after the seventieth year, is the rule. Arteriosclerosis is equally distributed between men and women. It is found in greater frequency in patients dying in general hospitals than in those dying in hospitals for the insane, which may be due to the greater number of arteriosclerotic cases in a general hospital, or to the possibility that prolonged stay in an asylum exercises a certain conservative action. In syphilitic psychoses the percentage of arteriosclerosis is apparently greater than in other psychoses. No definite difference in the incidence between dementia

praecox and manic-depressive psychoses has been established. The prevailing impression that there is a connection between arteriosclerosis and manic-depressive psychoses, rests on a purely empiric basis.

HART, Greenwich, Conn.

THE CLINICAL SIGNIFICANCE OF THE GRASPING REFLEX. E. A. FEODOROVA, Rev. Neurol. Psychiat. & Reflex. (Leningrad) **4**:151, 1929.

The author reviews the literature concerning the grasping reflex and states that the exact mechanism of the reflex is not clearly understood. Six neurologic cases are reported, with complete histories and details of examinations. The author is especially interested in the significance of the reflex in brain tumors. His conclusions are: The grasping reflex is an important symptom of involvement of the frontal lobes. In spite of the assertion of British authors, the upper frontal convolution is not necessarily involved. The subcortical ganglia are not necessarily involved. In marked increase of intracranial pressure the grasping reflex, even if it is found only on one side, has no localizing significance and is accounted for by edema of the ventricles.

KASANIN, Boston.

THE CONCEPT OF "EMOTIONAL AGE" AND ITS MEASUREMENT. C. O. WEBER, J. Abnorm. Psychol. **24**:466 (Jan.-March) 1930.

In this plea for the recognition of the validity of "emotional age," the author points out that lack of affective insight is quite as important as lack of intellectual orientation. Since the intellect is but the slave of the emotions, the attainment of emotional maturity is as essential as the advent of intellectual maturity. The author studies classes of children, who are asked about their emotional reactions, as for instance fear of thunder or love of collecting. The percentage of children in each group which shows this emotion, is plotted against the age in year and curves are thus established. The fastigium of the curve is taken as the normal age for the emotion being studied. Individual children are then studied according to their approximation to the standard thus established. The author hopes that this examination will reach the diagnostic importance of the intelligence test.

DAVIDSON, Philadelphia.

DERMOGRAPHIA IN THE INSANE. A. W. H. SMITH, J. Ment. Sc. **75**:458 (July) 1929.

From a study of 811 cases, including 241 females and 570 males, of epilepsy, primary dementia (dementia praecox), acute and chronic mania, melancholia, delusional and confusional psychoses, feeblemindedness, secondary dementia, senile dementia, dementia paralytica and encephalitis, Smith concludes that dementia praecox is the only class in which the percentage of positive results (10) is sufficiently above the average to warrant claims that dermographia is a characteristic phenomenon. He points out further, however, that the percentage in this condition is so small as to preclude a suggestion that it is in any way pathognomonic. Though the condition occurs more frequently in dementia praecox, the most conspicuous examples were observed in cases belonging in other classes, and the sign has therefore no value in diagnosis. It is more common in women than in men.

SINGER, Chicago.

A CASE OF FIBROUS MENINGIOMA (PSAMMOMA) IN THE CEREBELLOPONTILE ANGLE. G. H. MONRAD-KROHN, Acta psychiat. et neurol. **4**:203, 1929.

This case report deals with a psammoma of the cerebellopontile angle, the clinical history of which is unusual. The initial symptoms were convulsive seizures of the left side of the face. At this time there was possibly a slight decrease in the sense of smell and slight hypesthesia over the left trigeminal area. Later, left facial weakness developed, and still later the eighth nerve was involved. This was followed by complete paralysis of the left seventh and eighth nerves. At opera-

tion a small tumor was found lying along side a large bundle of nerves, probably the seventh and eighth. The patient made a good recovery except for paralysis of the eighth nerve.

PEARSON, Philadelphia.

DIAGNOSIS OF INCREASED INTRACRANIAL PRESSURE BY STUDYING THE RETINAL BLOOD PRESSURE. C. BERENS, Ann. d'ocul. **166**:853 (Nov.) 1929.

Berens refers to the relation of the cerebral to the retinal circulation and to the effect of spinal puncture on intracranial pressure as indicated by diastolic pressure in the retinal arteries, and he attempts to explain the apparent increase in intracranial pressure noted in some cases. The average pressure in the retinal arteries, as determined by several observers, is presented in a table which shows that the diastolic pressure obtained by Baillart and Seidel, with their respective methods, is substantially the same. He concludes that the Baillart method is the most practical clinical method of determining retinal blood pressure, and that by using tested instruments valuable data may be obtained in regard to the intracranial pressure. If the diastolic pressure in the retinal arteries repeatedly surpasses, by 15 mm. of mercury, half the brachial diastolic pressure, increased intracranial pressure may be suspected in the absence of disease of the retinal arteries.

CONOLLY, New York.

DEVELOPMENTAL ANOMALIES IN THE CEREBELLUMS OF EMBRYOS. JOSEPH C. YASKIN, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **31**:13, 1929.

Ten embryos, varying from 85 to 210 mm., were examined. A large series of anomalies in the cerebellum were found in these embryos although they were much older than those that had been studied by previous observers. The most important anomaly was the persistence of a canal lined with ependyma; the next important one was a median and paramedian collection of germinal cells, and finally, if not true cysts, at least a change in the ventral side of the cerebellar lobe in the nature of a zona spongiosa—a precursor of fetal cysts. In addition to these, there were also found in the fetal cerebellums hemorrhages which had a peculiar localization and which could well be regarded as the cause of later malformations.

KESCHNER, New York.

ERYTHROCYANOSIS CUTIS SYMMETRICA. E. V. KISTIAKOVSKY, Arch. Dermat. & Syph. **20**:780 (Dec.) 1929.

The disease called erythrocyanosis cutis symmetrica is defined and delimited by Kistiakovsky, who cites ten of his own cases and reviews the literature on the subject. He believes that the disease is due to endocrine dysfunction, citing as evidence the fact that it is so often associated with menstrual and ovarian disorders in women, and with thyroid and testicular disturbances in men. The fact that the red patches are inconstant in size and location is evidence that the disease is a vasomotor disturbance. Kistiakovsky considers the possibility of cold, tuberculosis, drugs and infection as etiologic agents, but concludes that the effect of disturbance in the endocrines on the vasomotor center represents the real cause. He suggests that pathologists call the disease "angioneurosis endocrinopathica polyglandularis." Endocrine therapy, the author concludes, is indicated.

DAVIDSON, Philadelphia.

EXOPHTHALMIC GOITER. INDICATIONS FOR STAGE OPERATION. JOHN DE J. PEMBERTON, Arch. Surg. **18**:735 (Feb.) 1929.

The progress of medical therapy in exophthalmic goiter has reduced markedly the incidence of cases in which a two-stage operation is indicated. Thus, in the Mayo Clinic in 1921, 13 per cent of the patients were treated with a multiple stage operation, whereas last year the number so managed was less than 0.5 per cent. A few indications for a stage operation remain—these are: cases refractory

to iodides and too severe for immediate radical thyroidectomy; cases with much tracheal compression, and cases which lead to serious dangers on the operating table.

DAVIDSON, Philadelphia.

DIFFUSION IN CEREBROSPINAL FLUID. CURRENT COMMENT, J. A. M. A. **94**:638 (March 1) 1930.

Doubt has recently been cast on the theory of a third circulation in the human system, viz., that of the cerebrospinal fluid. There are two reasons: (1) inconclusive experiments and (2) lack of purpose for such a circulation. Diffusion, it has been suggested, would meet all the physiologic requirements of the nervous system. Research conducted at the Washington University School of Medicine has borne this out. The dye diffused as rapidly in vitro as it did along the margins of exposed spinal, cerebral and cerebellar dura in dogs. The clinical importance of this fact is that therapeutic serums will reach all parts of the nervous system much more readily when given into the ventricles or by cistern puncture rather than by lumbar puncture. Furthermore, drainage can be much more safely done by cisternal than by lumbar punctures.

CHAMBERS, Syracuse, N. Y.

THE EXTENT OF THE FIELD OF VISION IN LENS MICROPSIA. ELLIS FREEMAN, Arch. Ophth. **3**:331 (March) 1930.

The apparent change in the size of visual objects in micropsia depends on physiologic factors rather than an actual decrease in the size of the retinal image. The explanation of Koster, and again of Jaensch, is given in detail. It had been supposed that micropsia was accompanied by a contraction of the visual field. For reasons of theoretic importance and also because it is well to understand all the modifications that concave lenses introduce into the world of the myopic, it was deemed advisable to determine by rigid experimental procedure whether concave lenses do extend the field of vision and perhaps thereby heighten peripheral acuity. The conclusions reached are that under the influence of a lens micropsia the field of vision does not change in the slightest degree.

SPAETH, Philadelphia.

OTITIC ABSCESS OF THE BRAIN. REPORT OF A CASE WITH SOME CONSIDERATION OF OPERATIVE INDICATIONS OF MASTOID INVOLVEMENTS. A. KOVACS, Arch. Otolaryng. **11**:438 (April) 1930.

As definite indications for operations are frequently wanting and as one is often unable to diagnose the site of an abscess, one should operate early and not wait for cardinal symptoms which lead to a diagnosis but which appear only when the case is too far advanced. All old cases of chronic purulent otitis should be viewed with suspicion. Patients infected with *Streptococcus mucosus* must be operated on early. Spinal puncture and roentgen examinations must be interpreted in conjunction with the clinical symptoms. The difficulty of interpreting sinus conditions, even with an inspection of the wall, is discussed by the author, and a case of temporosphenoidal abscess is reported to illustrate his view.

HUNTER, Philadelphia.

HEPATO-LENTICULAR DEGENERATION: A FINAL NOTE. STANLEY BARNES and E. WESTON HURST, Brain **52**:1 (April) 1929.

Barnes and Hurst publish the final clinical data and complete autopsy observations of the fourth and last case of their series of hepato-lenticular degeneration, first reported in 1924. Tetanoid convulsions occurred in this patient and in one of the previously reported cases. A possible cause for this type of spasm, offered by the authors, is a flooding of the central nervous system with toxins, which cannot be destroyed because of the extensive destruction of the liver.

BECK, Buffalo, N. Y.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Feb. 20, 1930

E. W. Taylor, M.D., *President, in the Chair*

A Case of External Hydrocephalus. Dr. Benjamin T. Burley.

A child, aged 11 months, whose head measured 21¾ inches (55 cm.), had a history of normal birth and infancy up to 6 months of age. Then there occurred a fairly rapid enlargement of the head, which at 9 months had reached 22½ inches (56 cm.) in circumference. During the process of enlargement there was some vomiting, internal strabismus, restlessness and mental dulness, but there had been no actual palsies.

I assumed charge of the case when the child was 9 months of age. At that time I removed 300 cc. of blood-stained fluid from the subdural space by a puncture through the open anterior fontanel, replacing this fluid with air and obtaining roentgenograms of the head showing an extensive area of air totally covering the surface of the brain, but not connected with the cerebroventricular system. The latter fact was proved by the injection of methylene blue (methylthionine chloride, U. S. P.) into the subdural space, and later finding by lumbar puncture, that the spinal fluid was perfectly clear with no blood or coloring elements. Later, ventriculograms were made by injection of air into the spinal column and these showed normal lateral ventricles with no connection with the extensive subdural space.

The case resembled hydrocephalus, although since the intracranial drainage, the child apparently has been perfectly well physically and mentally and now weighs 26 pounds (11.8 Kg.). There was no history of trauma, and the gradual onset seems atypical of hemorrhagic pachymeningitis. The bloody fluid is also atypical of hydrocephalus. It is the combination of symptoms that makes this case of rather uncommon interest.

DISCUSSION

Dr. Paul I. Yakovlev: What is the probable future of this patient?

Dr. T. J. Putnam: Is it not possible that this is a case of subdural hematoma that has become cystic rather than a true hydrocephalus? Such cases have been reported in children of this age.

Dr. C. A. MacDonald: Were the fundi normal, and were there any other eye symptoms?

Dr. E. W. Taylor: Was the patient normal mentally? His appearance was normal as presented here.

Dr. D. Gregg: Has the head continued to enlarge?

Dr. B. T. Burley: I do not know what the patient's future will be. He seems to be doing well at present. I think that it is possible that this is a subdural accumulation of fluid. The optic nerve heads were normal. The patient was normal mentally until the age of 5 months, when evidence of retardation was noticed. Since tapping the head the mental condition has improved, and the head has grown somewhat smaller. I presented this case for help in diagnosis, and I should like to ask Dr. Horrax if he has had any similar cases.

Dr. G. Horrax: I have seen similar cases at the Children's Hospital rather superficially. The distention of the head has subsided after repeated tappings, but the precise mechanism of the process is obscure.

NEW YORK NEUROLOGICAL SOCIETY AND NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY

Joint Meeting, March 11, 1930

LOUIS CASAMAJOR, M.D., *President, New York Neurological Society, in the Chair*

GROSS ANATOMY OF THE NEOSTRIATUM. DR. WALTER M. KRAUS and DR. W. A. HORWITZ.

Customarily, the neostriatum is described as consisting of two independent structures, the caudate nucleus and the putamen, but in reality these two are one nuclear mass. Reconstructions of the corpus striatum of a series of mammals, e. g., opossum, cat, monkey and man, were undertaken with the view of determining the morphology, especially of the neostriatum. Serial sections of these animals, cut from 50 to 100 microns in thickness and stained by the Loyez-Weil method, were used. Beginning at the most anterior portion of the caudate nucleus, every tenth section was taken and projected ten times its normal size; tracings of the striatal areas were made. Wax plates of 5 cm. in length and 1 cm. in thickness were cut, corresponding to the striatal nuclei. These sections were superimposed on each other, and the result was a reconstruction of the caudate-putamen mass, ten times the size of the original nucleus.

Comparing our models with the description given in the standard textbooks on anatomy, we find certain differences. The impression conveyed by the books is that the caudate and putamen are two independent bodies which are fused anteriorly, but posteriorly are independent. Tilney and Riley, in "Form and Functions of the Nervous System," said that "the tail of the caudate nucleus swings around the temporal horn of the lateral ventricle and ends in connection with the nucleus amygdalus." They did not mention that there is a fusion posteriorly, as well as anteriorly, of the caudate-putamen. They also did not mention that there are accessory nuclear bridges all the way around connecting the caudate with the putamen in the middle and posteriorly as well as anteriorly. They noted merely the occurrence of these internuclear bridges between the head of the caudate nucleus and the lentiform nucleus in the region of the anterior limb of the internal capsule.

Cunningham, in his "Textbook of Anatomy" (p. 638), stated that "when a section is made further back (posterior to the anterior commissure) the divided lentiform nucleus is seen to be completely cut off from the caudate nucleus by the internal capsule." He also gave the impression that the tail of the caudate ends only in the amygdaloid nucleus.

The same impression of the relationship of these two structures is conveyed by other anatomists — Ranson, Villiger and Toldt.

From our investigations, as well as from the earlier work of Hausman, it appears that these two structures are really one nuclear mass that is pierced by the internal capsule, and that posteriorly as well as anteriorly the caudate-putamen portions are fused.

It is interesting to note in connection with the penetration of a nuclear mass by a fiber projection system that this conforms to the view of Kraus and English that pathways within the brain when first laid down take the shortest course from their nucleus of origin to their nucleus of destination, subject to the displacement by the cavities of the brain.

In studying these various cross-sections, we were also impressed with the fact that there are masses of nuclear material bridging the head, body and tail of the caudate and the putamen. These are found at all levels and not only in the anterior portion. At a level just posterior to the putamen these isolated

nuclear masses are closely concentrated; as one proceeds further posteriorly, they are seen to diverge fanwise to meet the wider arc of the tail of the caudate.

In a lower form of mammal, the opossum, these facts are brought out more clearly. In the opossum the internal capsule is not as well developed and appears as only a slight strand between the caudate and the putamen, which is plainly fused posteriorly as well as anteriorly. In man, in whom the capsule is more developed, the separation is greater, especially in the posterior portion.

In the cat and monkey, as well as in the opossum and man, the internuclear masses are seen at all levels connecting the two portions of the caudate-putamen nuclear mass. In all of these animals the tail of the caudate can be followed posteriorly into the putamen as well as to the amygdaloid nucleus.

DISCUSSION

DR. LOUIS HAUSMAN: The point which Dr. Kraus has made is of considerable interest, because among other things it emphasizes the inadequacy and inaccuracy of the term "lenticular nucleus." This is an unfortunate designation as it links the putamen with the globus pallidus, and does not recognize the fact that the putamen is part of the neostriatum and belongs with the caudate nucleus. This relationship is well established phylogenetically; in the fish and in the frog the globus pallidus is the only part of the corpus striatum present and is identified as the paleostriatum. The neostriatum first appears in the reptilian series. There it exists as one mass, delimited from the paleostriatum by a white lamina and fissure. This separation of neostriatum from paleostriatum is also well indicated in the bird series, by the white lamina, the striate artery and the paleoneostriatal fissure. In these animals the division of the neostriatum into its two nuclei, i.e., putamen and caudate, is not well defined, due to the absence of an internal capsule. The separation of putamen and caudate, which first appears in mammals, is really caused by the accidental passing of the internal capsule through the corpus striatum, giving the impression that the putamen belongs to the globus pallidus rather than to the caudate. In the reptilian series there is no such separation because there is no internal capsule to split the striatal mass, except in *Iguana*, in which there are a few fibers passing through the corpus striatum and suggesting the division into putamen and caudate. In the bird there is no internal capsule, and hence no such division of the neostriatum.

Of considerable interest is a case in a human being reported by Dejerine and quoted by Kappers, in which the putamen and caudate were more or less fused. The case was one of cortical atrophy, with a corresponding reduction in the volume of the internal capsule, so that there was no division of the neostriatum.

In addition to the phylogenetic evidence, there are also histologic, physiologic and embryologic facts to support the view that the putamen and caudate should be considered together and apart from the globus pallidus. The latter, for example, myelinates about the fifth or sixth month of fetal life, whereas the neostriatum myelinates later. The point which Dr. Kraus emphasizes is one which stands out clearly in sagittal Weigert-stained sections of the forebrain, in which the tail of the caudate can be clearly seen swinging around and under the putamen to enter the latter in the form of a hook. It is curious that this well defined anatomic relationship is overlooked in most textbooks on neuro-anatomy, in which the authors prefer to present the tail of the caudate entering the amygdaloid nucleus. I wish to ask whether in Dr. Kraus' studies of the mammalian series he noticed any connections between the corpus striatum and the cerebral cortex. Today this is a much disputed point. One series of workers, represented by Wilson and Cajal, maintains that there are no such connections; opposing this view is another group, represented by Dejerine, Flechsig, Minkowski and others. It would be interesting to hear what Dr. Kraus has found in this connection.

DR. LOUIS CASAMAJOR: This presentation has been of considerable interest to me. Some years ago, Dr. Tilney and I made a series of models of the entire

striate body, and our observations exactly coincide with those of Dr. Kraus. The caudate is really a part of the putamen, and, as Dr. Kraus says, it is only split off accidentally by the fact that the internal capsule fibers and others of the same group pass through it. It is purely accidental that it should be divided into two nuclei, and one must look on it as one structure. Certainly phylogenetically, ontogenetically and structurally it must be one body.

DR. WALTER M. KRAUS: In answer to Dr. Hausman's question, we have found no relation between the cerebral cortex and the corpus striatum.

THE ORIGIN AND FORMATION OF THE SENILE PLAQUE. DR. ARMANDO FERRARO.

This article will be published in full in a later issue of the ARCHIVES.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

March 20, 1930

E. W. TAYLOR, M.D., *President, in the Chair*

A COMPARISON OF TREATED AND UNTREATED CASES OF DEMENTIA PARALYTICA. DR. JULIUS LOWMAN.

From a study made at the Boston State Hospital of treated and untreated patients with dementia paralytica over a nine year period from 1921 to 1929, the following comparisons and conclusions were made: (1) In 166 treated cases there were 20 per cent remissions. (2) In 209 untreated cases there were 8.1 per cent remissions. (3) If the cases in which so little treatment was given and in which the remissions were probably independent of the treatment are excluded, the remission comparison would be 14 against 8.1 per cent. (4) The selection of the cases for treatment would alter such a difference still more, for it was found that many more *a priori* hopeful cases were included in the treated than in the untreated group. (5) The majority of remissions in the treated series followed treatment that was begun within a few months after the onset of the psychosis. (6) There was no relationship between the amount of treatment and the incidence and the length of remissions. (7) Although there were more remissions and more patients living for more than five years in the treated group, the advantage in duration over the patients in the untreated series was only one year. The average length of the disease in the former group was 3.3 years and in the latter group 2.2 years. (8) The percentage of remissions in the treated series is decidedly below most of those reported by workers using either malaria, tryparsamide or milk. However, it is possible that the selection of cases used by each worker accounts, in great measure, for such differences in results.

BASIC CONSIDERATIONS OF THE PHYSIOLOGIC APPROACH TO THE PSYCHOSES, ESPECIALLY IN RELATION TO CERTAIN PSYCHONEUROSES. DR. ABRAHAM MYERSON.

The starting point of this formulation is that man is an organic fragment of an organic universe, that everything that happens to him is, strictly speaking, organic. Whether the human being reacts to a toxic influence or to the words or looks of others, the stimulus in its last analysis is organic, and the response throughout the organism is organic. Thus, the separation into functional and organic is, strictly speaking, illogical, although it may be useful. There is no fundamental difference between the vasovisceral, motor, endocrinal and cerebral changes produced by the drug, atropine, and the state of fear. There is no reason to say that the dilated pupils, the dry mouth, the increased heart rate, the increased respiration, the pale face, the trembling limbs, the increased urination, and even the involuntary bowel movements of fear are in any different category from the effects of a drug or of some infection. In other words, one may study the psychoses and psychoneuroses from the standpoint of their physiologic reverberations, as well as from the standpoint of those so-called psychologic situations from which

they arise or are said to arise. Moreover, the psychologic events are open to the difficulty that their relationship as cause is subject to doubt. They cannot be reproduced and are not accessible to scientific investigation in the usual sense, and at the present time they have to be symbolized and interpreted by an elaborate and, in my opinion, arbitrary technic.

I have followed this formulation as the leading approach to the researches which are here cited and also as the main part of my present approach to a certain group of the psychoneuroses. This group of the psychoneuroses, however officially classified, is marked by great physiologic phenomena, although it is true that these phenomena may also be interpreted as psychologic if one cares to separate the world of inner events in that way.

The patients suffering from the state in question show: (1) disturbances in the physiology of nutrition in that they lack appetite and often have shown nausea and vomiting; (2) disturbance in the mechanism for recuperation in that they are unable to sleep; (3) disturbance frequently in the physiology of reproduction — the males become partly or wholly impotent and the females show anomalies in menstruation, often developing into amenorrhea. There is also disturbance in the discharge of energy in that the patients are incapable of sustained activity in any field, physical or mental. This may be so extreme as to force them to bed, where they remain for a long time.

Certain paresthesias are complained of which, in my opinion, are as genuine as the paresthesias of the organic diseases and are to be explained on some physiologic basis. These are the paresthesias of the head, neck and mouth, the paresthesias of the throat, erroneously called globus hystericus; paresthesia in the epigastrium which often leads to all kinds of gastro-intestinal intervention and treatment; paresthesia of the legs and arms, which leads to the pulling of teeth and the enucleation of tonsils on some vague notion of a focal infection. There are very important paresthesias of the special senses. The patients complain of spots and blurring of the eyes, of tinnitus, of a bad taste in the mouth which is organic, as evidenced by a dried, furred tongue. They often have paresthesias of the sense of smell and complaints which are related to disturbances in touch and peculiar sensations are legion: burning sensations, hot and cold feelings, etc.

One often sees, associated with the paresthesias, definite twitchings that are objective, in that the examiner may see them, and which correspond to some extent to the complaints of the patient.

I have, in my own mind, divided the paresthesias of these patients into groups, believing that they come in large measure from what might be called the flow of inner impulses, cenesthesia. They are arranged in three groups: (1) Paracenesthesia, abnormal sensations. (2) Hypercenesthesia, in which sensations from the heart, lung or gastro-intestinal tract become definitely discernible to the patient. (3) Anacenesthesia; in the extreme psychoneuroses this state becomes apparent in that the patients feel dead, things become unfamiliar and a sense of unreality on the subjective side is complained of.

Necessarily, this physiologic formulation is only a hypothesis at the present stage of understanding of the psychoneuroses. I believe, however, that when therapeutics are organized on the basis of these situations, one has to do remarkably little psychologic treatment or so-called psychotherapy. I see no reason to believe that a man's mood cannot be reintegrated by physical agents. The change of mood which is caused by alcohol, tea, coffee, food, sleep and other physical agents indicates that one can approach the human being's mind and emotional state through his body.

My procedure, simply put, is to outline the situation as I see it to the patient if he is capable of understanding such a formulation; to put him under such physical conditions of rest, exercise, physical therapy and drugs as tend to reestablish appetite and sleep. If a man can eat and sleep, he can usually handle his own complexes better than they can be handled by me. I am not yet ready to concede that psychologists and neurologists are wise enough to direct the intimate psychologic, social and moral reactions of other people.

SEDIMENTATION RATES IN VARIOUS PSYCHOSES. CAROLINE STEPHENSON.

This is a brief discussion of the sedimentation rate, history and importance of the rate in various conditions, with a comparison of various methods and results obtained by other investigators, particularly in psychiatry. I have paid especial attention to the modification of the heparin method described by Rourke and Ernstene, which was the method used in observations at the Boston State Hospital. The results on rates determined on about 110 patients showing various psychoses were as follows: There was no significant difference in rates on blood from the carotid artery, jugular vein, brachial artery and basilic vein taken from the same patients at the same time; patients with dementia paralytica showed 77 per cent increased rates, not dependent on the amount of deterioration or stage of the disease; manic-depressive patients showed increased rates; patients with dementia praecox showed varying rates not dependent on the amount of mental deterioration nor consistently on types; the simple type showed normal rates, the catatonic type increased rates and the hebephrenic and paranoid types showed varying results; about 60 per cent of each had increased rates; these patients had no apparent abnormal condition other than dementia praecox.

THE WHITE BLOOD CELLS IN DEMENTIA PRAECOX AND DEMENTIA PARALYTICA. DR. WILLIAM DAMESHEK.

The white blood cells are derived from three systems: the bone-marrow, which produces the polymorphonuclear cells; the lymphoid tissue, which produces the lymphocytes, and the reticulo-endothelial tissue, which produces the monocytes. It was thought that these systems could be observed clinically by careful study of the blood smear. Cover slip preparations were used; from 250 to 500 white cells were counted; note was made of: the presence of immature forms of polymorphonuclear cells (according to the Schilling method); "toxic" changes in the polymorphonuclear cells; plasma cells; histiocytes, etc. There were no essential differences in the total white cell counts or their relative proportions in the blood from various situations (brachial, basilic, carotid, jugular, finger or ear).

In seventy cases of dementia praecox, normal conditions were present in 70 per cent. In 30 per cent there was a triad of low polymorphonuclear count, lymphocytosis and an eosinophilia of over 4 per cent. This occurred especially in the catatonic cases. Eosinophilia up to 20 per cent occurred in about 10 per cent of the cases.

In seventy cases of dementia paralytica a monocytosis of over 10 per cent occurred in 52 per cent; histiocytes were present in 20 per cent; plasma cells in 27 per cent; toxic changes in 80 per cent, and an increase in immature forms of polymorphonuclears in 12 per cent. The appearance of histiocytes and plasma cells is linked with the pathology of dementia paralytica. In one remarkable case in which several convulsions occurred each night, there was a monocytosis of 40 per cent or more with the appearance of a large number of histiocytes, many containing phagocytosed particles and vacuoles. These occurred with convulsions and disappeared when convulsions were stopped, to recur when convulsions again started. It was thought that the convulsions in some way caused the discharge of monocytes and histiocytes from the abnormal tissue in the brain.

No definite criteria as to diagnosis or prognosis could be found from the study of the white blood cells.

BIOCHEMICAL STUDIES OF ACTIVITIES OF THE BRAIN BY THE JUGULAR PUNCTURE METHOD, WITH SPECIAL REFERENCE TO CARBOHYDRATE AND GASEOUS CONTENT. DR. A. MYERSON and DR. R. D. HALLORAN.

In 1927, puncture of the internal jugular vein, a new technic, was added to puncture of the carotid artery, a known technic, for the biochemical study of the blood entering and leaving the brain. It seemed logical that such a study might reveal any changes incident to the passage of the blood through the brain.

Lumbar puncture was included in order that the only other fluid carrying metabolic products of the brain might be tested.

Well advanced cases of dementia paralytica and dementia praecox were selected for tests as representative of the known organic and the unknown, so-called functional, diseases.

The internal jugular vein, vagus nerve and internal carotid artery lie in close relation to each other just inside the mastoid tip and under the anterior border of the sternocleidomastoid muscle throughout their course in the neck, the internal jugular vein being the most external and the nerve lying between and posterior to the artery and vein. The common carotid artery branches into the internal and external carotid arteries at about the level of the hyoid bone.

Under procaine hydrochloride local anesthesia, the internal jugular vein is punctured with a 25 cc. Luer syringe and a 1½ inch (2 cm.) 19 gage needle thrust its full length perpendicularly to the skin just inside and below the mastoid tip. The vein receives no tributaries from the point of exit from the brain until lower down in the neck. The internal carotid artery may be entered in a similar manner slightly lower in the neck, and about 2 mm. mesial to the point of entry of the internal jugular vein. As this performance is difficult, the common carotid artery is generally used. It is entered at about the level of the hyoid bone with the needle pointing upward parallel to the artery, and the barrel of the syringe at about a 30 degree angle with the skin, the pulsation acting as a guide. Blood from a vein and an artery in the arm is taken for comparison. Simultaneous or nearly simultaneous punctures of the carotid and brachial arteries and of the internal jugular and basilic veins are made by two operators to eliminate the possible influence of altered mental states between the punctures. All bloods are drawn into oxalated syringes with the exception of those to be tested for calcium. Air is excluded from specimens tested for carbon dioxide, oxygen and chlorides by placing mineral oil in the syringe barrels, and passing the blood under oil into a collecting tube.

Exploratory tests were made for sugar, cholesterol, fatty acids, lecithin, urea, nonprotein nitrogen, phosphates, chlorides, calcium, carbon dioxide and oxygen. Because the only essential difference found thus far between the blood of the brain and that of the arm occurred in sugar, carbon dioxide and oxygen content, seventy-five carefully checked cases of dementia praecox and dementia paralytica were examined for these products. The blood of a few epileptic patients and of apparently physically and mentally normal employees was similarly tested for controls. Studies of the carbon dioxide and oxygen are not conclusive, as this work is still in its early stages. Specific gravities were estimated on the blood from the various locations.

Tentative conclusions were reached on the basis of this work: (1) That the brain is an active metabolic organ is now a matter of fact rather than of supposition. (2) There is as great and probably a greater disappearance of sugar in the passage of the blood through the brain than in its passage through the arm, as evidenced by the consistently lower sugar in the internal jugular vein than in the basilic vein blood. (3) This is probably a physiologic rather than a pathologic phenomenon. (4) There is evidence that the sugar content of the carotid and brachial arteries is the same or nearly the same. (5) There is an equal and possibly higher carbon dioxide and lower oxygen content in the venous blood from the brain than in that from the arm. (6) Observations with other substances are not complete, but thus far no consistent differences have been noted between the blood entering and leaving the brain, and that entering and leaving the arm. (7) Differences between the blood sugar content in the various locations in the neck and arm are unaccompanied by differences in weight. (8) This method of study of the metabolism of the brain is practicable and without danger to the patient. (9) It constitutes a new and logical approach to the study of conditions of the brain in health and disease.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, March 20, 1930

A. B. YUDELSON, M.D., *Vice-President, in the Chair*

SHALL HYPNOTICS BE USED IN THE TREATMENT FOR INSOMNIA IN PSYCHONEUROSES? DR. MEYER SOLOMON.

By hypnotics, I refer in particular to drugs of the barbital group, much less to the sedative group (bromides, chloral and paraldehyde) and not at all to the so-called euphorotics (alcohol, opium, morphine, heroin and cocaine). I shall consider also psychogenic insomnia, occurring in the psychoneuroses, alone. The dominant current attitudes of the medical profession are: 1. No drug treatment, including hypnotics, is to be used in these conditions. 2. Hypnotics are to be used only as a last resort. 3. They are to be used only for a brief period and only after other measures have failed, or if the insomnia is so pronounced that immediate relief is necessary, or both.

Most physicians hesitate to admit that they prescribe hypnotics under any circumstance whatever. Many patients and families fear the use of hypnotic drugs, which they refer to as "dope." The main arguments against this treatment are: 1. It does not affect the cause. 2. There are possible toxic effects. 3. There is danger of drug addiction. 4. There is danger of self-medication. 5. A suicidal use is possible. 6. The effect is temporary. 7. It is an artificial crutch. 8. It is a line of least resistance, an easy way out. 9. Other necessary treatment may be neglected by the physician, the patient or both. 10. It gives the patient the wrong idea of the causes and of the fundamentally important psychologic treatment required. These objections apply to all palliative drug therapy and carry no weight if the drugs, including hypnotics, are used judiciously. Although I agree that, in cases in which the cause of sleeplessness is psychic, the primary object is to ascertain all possible causes of mental discomfort, nevertheless, a more or less prolonged psychologic investigation may be necessary and may take some time; in the meantime the patient needs sleep.

In many instances, the original cause or causes of anxiety are no longer operative, and the patient may be worried mainly about symptoms, including insomnia; or both the original and the present causes of anxiety need to be considered. A fundamental principal in therapy should be that patients must be satisfied and relieved, if possible, by nondangerous methods adapted to the particular case.

The courses open to a physician are: (1) never use hypnotics; (2) use them promiscuously and unjudiciously; (3) use them judiciously, with or without causative treatment. I favor their judicious use, combined with causative psychologic therapy and other measures indicated (physical therapy, etc.). There are harmful effects, physical and psychologic, direct and indirect, of prolonged insomnia in psychoneuroses. I believe that a short period of sleep, in addition to having direct benefits, helps to break the habit of sleeplessness, diminishes or eliminates other undesirable physical and psychologic effects and puts the patient in a condition that renders him more amenable to suggestion, explanation, analysis and other psychotherapeutic procedures. The patient must be satisfied that he is sleeping soundly and sufficiently.

I do not advocate that every psychoneurotic patient, or even that every psychoneurotic patient with restless sleep, should be given hypnotics. I do insist that one can be as unscientific in withholding hypnotics as in giving them when they are not necessary; as a temporary measure it may be absolutely necessary to give them.

Concerning technic and precautions, one should obtain the patient's views on insomnia and hypnotics, give a full explanation to him concerning the causes of his sleeplessness, relieve groundless fears, and, if it is decided to use hypnotics temporarily, explain that this is in addition to the primary causative psychotherapy. The first dose should be large enough to be effective. After a sufficient number

of nights of good sleep have been obtained, usually not later than after two or three weeks, the dose should be reduced with the patient's knowledge until gradually he is taking none. The hypnotic may be varied when necessary during this period. Whether or not the patient should know what drug he is getting varies with the case. Although, in most instances, the prescription can be given to the patient or a relative without much fear of abuse, it is generally best that he should not know the name or dose of the drug, other than that he is being given a hypnotic. Hypnotics that do not create a craving should be used.

FEAR AS A BASIS FOR MENTAL DISORDERS. DR. RALPH C. HAMILL.

Fear is the underlying factor in all forms of mental disease and gives the pathologic quality to mental states. Tracing the evolution of fear, it is seen in its simplest and most unmistakable form in the night terrors of children. Up to the age of 5, a common picture is that of being eaten by animals. In the next few years the fear shows a change toward resentment. A boy, aged 7, spoke of a fear that a piece of iron was lodged in his mouth, the outcome of a dream of a bit in a pony's mouth — a symbol of restraint. Another, aged 8, a bedwetter, had dreams of robbers — his parents — who came into his room and intended to kill his mother. Such dreams arise from a sense of inadequacy and resentment against control, which also cause thoughts of revenge in the form of killing the parents. The most pertinent example of this reaction of the young is contained in the folk story of Jack and the Beanstalk. The ogre is the father — a giant as compared with the child — from whom the son steals and who is finally killed.

At the age of puberty, new elements, those of sex, begin to be added. It is my opinion that children realize the truth of sex only when the changes of puberty make them physically able to appreciate its actuality. In the child's attitude toward this situation, the parents may be made to appear, more or less symbolically, ridiculous in their sexual conduct, or they may appear despicable and filthy. Women often experience a reaction to sexual feelings that consists of a change from feelings of passion to those of depression; an understanding of this seems to me essential for the understanding of all those states in which depression plays a part. Fear may also change acute physical sexual pleasure into pain, as in many cases of so-called pain in the genital and pelvic region of women, especially such as vaginismus and coccygodinia. Mankind is played on by two forces — animal impulses and social ideals — opposite in aim, which between them can produce a sense of inadequacy, feelings of inferiority, fear, resentment, etc.

Concerning the manifestations of fear in the different mental diseases, the simplest is merely another name for fear — nervousness. In a boy, aged 12, "tubular vision" developed as a method of not seeing things other than those he was looking at directly. The deliria are associated with terrifying paresthesias. Every patient with melancholia seems to suffer with some sort of guilty feelings. Is not the suffering in melancholia as clearly purposeful and self-inflicted as that of the religious martyrs and flagellants? Persecutory delusions are obviously fears. Ideas of reference are due to a fear of responsibility. Egocentric ideas are a defense against the long process of corrective upbringing of childhood. In dementia paralytica there is a damage to function by the disease, but the ideas of punishment produce a picture of hypochondria or depression, and reactions against ideas of inferiority and inadequacy produce the grandiose delusions.

DISCUSSION

DR. MEYER SOLOMON: I recall that Boris Sidis, in a book entitled "The Causation and Treatment of Psychopathic Disease," endeavored to prove that in all instances the psychoneuroses are due to anxiety and fear, and that the latter are offshoots of the instinct of self-preservation. In the World War it was proved that fear played the greatest part in the production of war psychoneuroses. Although fear may be centered about the sexual impulse, it may also be centered about other motivations. In the psychoneuroses in the average adult, self-

preservation, centered about ambitions and unfulfilled yearnings with resultant anxiety and fear, plays a great part; the same is true in the child with his yearnings and unfulfilled wishes. I believe that with increasing interest in the study of the personality of children, guidance of children and the education of parents, the unnecessary fears and feelings of inadequacy that grow with the years will be eliminated more and more.

DR. LEWIS J. POLLOCK: I have been interested in a phase of fear that is perhaps not directly related to Dr. Hamill's thesis. It is not strange that similar trends may be found in dissimilar psychoneuroses. The interpretations that are put on these neuroses bear the same relation to one another as those which occur in investigative work in physiology in which similar observations may lead to different conclusions by different investigators. The classification of fears in relation to prognosis has interested me. Some types appear in cases usually designated as psychoneuroses, and others in cases that later develop into frank psychoses. Certain simple, purely logical fears are common in the neuroses. One of the most common is, perhaps, the fear of death. Others are the fear of creating a scene, losing control of oneself or losing one's mind. Fears of this type are common and may be amenable to explanation, but on occasions they are an expression of a psychosis. On the other hand, many fears are more bizarre. Mysophobia occurs often and becomes well fixed, and all explanations fail to induce the patient to acquire another point of view. In these patients, the mechanisms of schizophrenia often develop, while in many others a psychosis never develops. I recall the case of a man who feared to leave his house because he might soil his trousers. This fear was associated with an uncomfortable experience which occurred when, as a youth, he was dining in a glittering restaurant. The fear cut him off from all social intercourse for many years. One explanation of the control of sphincteric muscles cured him of this fear, and he has led a normal life for many years. On the other hand, a fear of poisoning by wood alcohol and by contagion has prevented the development of a case of frank schizophrenia. Another difficulty is that of separating the fears of an anxiety neurosis from those of melancholia. The content of the fears may be exactly the same, but in the latter case no amount of explanation or persuasion can dissipate them.

DR. A. B. YUDELSON: Dr. Hamill's paper brought to my mind the case of a woman, aged 26, who was playing leading rôles in Shakespearean plays. For two and a half years she suffered from depression which she experienced when the climax of a play was about to come. She suffered from constriction of the vocal cords, as if some one had caught her by the throat. This did not prevent her from going through the climax, but she was exhausted and shaken up at the end of the play. Repeated examinations made by competent laryngologists did not disclose any local anatomic disturbance or abnormality. When I examined her after one of the most severe paroxysms she had had in a long time, I found that, at about the age of 7, when she was first initiated into Sunday school, she liked to listen to the stories; after a while they became uninteresting, and she preferred to stay at home on Sundays. To accomplish this desire she had to resort to subterfuge. She had a sister, aged 13, who suffered from "dyspepsia" and was kept in bed on Sundays to rest, while the younger child had to go to Sunday school. She finally decided to play a trick on her mother. She thrust the handle of a teaspoon down her throat, making a retching noise as if about to vomit. This made the mother fear that the patient was going to have "dyspepsia." Henceforth she was kept in bed on Sundays. However, she suffered remorse. Some years later, at communion (she was an Anglican Catholic) she thought of the deception. She had forgotten about the episode until she qualified for the stage, when she began to suffer with this trouble. When questioned as to whether she still suffered from a feeling of guilt because she had deceived her mother she said that she had not thought of it since the day of her confirmation when she felt sorry that she had deceived her mother. It was explained to her that the choking sensation she experienced on the stage was a form of fear from

which she suffered as a result of the hidden guilt. Since then she has not experienced any further constriction of the throat.

DR. R. C. HAMILL (closing): I should like to spend more time in the matter of the evolution of what we call psychotic or mental states from the conditions of childhood. Fear that underlies behavior problems of early childhood evolves into resentment. Resentment leads to antagonisms, especially against parents. Ideas of revenge or of violence give rise to feelings of guilt; the persistence of such feelings helps to develop feelings of inadequacy. Dr. Pollock spoke of such ideas perhaps disguising and finally uncovering melancholia. After all, melancholia is merely a name; it does not represent anything concrete. Fear, guilt and a sense of inadequacy vary in degree. When active, they may cause great depression. A state of melancholia then exists.

Many more of the children who are brought to me for treatment are from the parochial than from the public schools.

I think that the condition referred to by Dr. Yudelson is to be spoken of under the term "inferiority." My thesis is that out of the fears of childhood come the signs of inadequacy and inferiority, and signs of guilt, that under difficulties of any sort may crop out later in life.

CHONDROMAS ARISING FROM THE INTERVERTEBRAL DISKS. DR. PAUL BUCY (by invitation).

In a case of chondroma arising from the intervertebral disk between the third and fourth lumbar vertebrae, the symptoms were those of involvement of the cauda equina. The lesion was localized by the use of iodized poppy seed oil 40 per cent. Following laminectomy and the removal of the tumor, the patient was completely relieved. Microscopic examination of the tumor revealed it to be a fibrocartilaginous neoplasm. A review of the meager literature on the subject revealed sixteen cases, including the one reported here. Nine were in the cervical region, three in the thoracic and three in the lumbar region. In one, the location was not specified. This indicates that the lesion is not limited to the cervical region. That all of these cases should have appeared in the short space of time since Stookey's original paper on the subject seems to indicate that this type of lesion has been frequently overlooked.

Lantern slides demonstrating the pathologic processes of this condition were presented.

DISCUSSION

DR. ARTHUR WEIL: This report is of interest because the histologic pictures showed features that also may be found in another kind of tumor arising from the intervertebral disks of this region, namely, the chordomas. The areas of myxomatous character, the physaliphorous cells and finally the fibroplastic structures may also be seen in chordomas. Charles Davison and I have described such a tumor at the third lumbar vertebra (Malignant Chordoma of the Lumbar Region, ARCH. NEUROL. & PSYCHIAT. **19**:415 [March] 1928), which at the onset of the disease showed the typical features of a chordoma. Nine months after the operation, the tumor had formed regional metastases, and the histologic picture had changed completely. In addition to the remnants of the original tumor, structures of the type of a myxochondroma were found intermingled with it. The question might be discussed whether chordomas, which arise with preference from the intervertebral disks at both ends of the spinal column, may not be stimulated by an operative procedure to grow at a more rapid pace while they undergo such histologic changes as those described.

A NOTE ON THE CELLS OF THE SPINAL ARACHNOID IN PATHOLOGIC CONDITIONS. DR. ISADORE B. DIAMOND.

This paper will be published in full in a later issue of the ARCHIVES.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, March 28, 1930

WILLIAMS B. CADWALADER, M.D., *President, in the Chair*

METASTATIC MELANOMA IN THE BRAIN. DR. ALFRED GORDON.

A woman, aged 45, had what was thought to be a gallbladder disease, six years before presentation; it was characterized by pain radiating from the right hypochondrium to the right shoulder. Since then, several similar paroxysms have occurred at long intervals. During the entire period there were evidences of pronounced gastro-intestinal upset, with dyspnea and headache. Two years before presentation, the right eye was enucleated, supposedly for a malignant tumor.

Physical examination revealed: an enlarged thyroid; a palpable mass in the right upper quadrant of the abdomen, about four finger breadths below the costal margin, which moved with respiration down the entire right flank reaching the right crest of the ileum, and an enlarged tender lymph gland on the left side of the neck just above the angle of the jaw. The surgical diagnosis at that time was a tumor of the liver. On the operating table the liver was found to be very large; the wound was closed as nothing could be done. Soon the patient developed an effusion in the right lung, and the temperature rose to 100 F.

Fibrillary twitchings soon developed in the left side of the face, and the face became deviated to the right. Rapid muscular contractions became generalized, but were especially marked on the left side of the body. Convulsive seizures, clonic in type, appeared in the left side of the body and on the right side of the face. The patient went into a status epilepticus. The eyes showed nystagmoid movements and turned strongly to the left. She became unconscious and died soon after.

Autopsy revealed that both adrenals were about ten times the normal size. Metastatic infiltrations were found in the thyroid, cardiac muscle, right auricle, diaphragm, both lungs, the lymph nodes, liver, pancreas, both kidneys, stomach, duodenum, uterus, mesenteric glands and the brain. There were small nodules in the parietal lobe, in the temporal lobe near the apex and in the right hemisphere, and one in the posterior portion of the right cerebellum. They were all of dark color and embedded in the cortex. The right hemisphere was congested. The left hemisphere was paler and presented laterally some exudate, especially at the sylvian fissure. At the base there was an extensive meningeal hemorrhage at the level of the cerebellum. An anteroposterior section of the brain shows a circular hemorrhage, the size of a half dollar, within the right basal ganglia, extending from the caudate nucleus back to the optic thalamus and involving the anterior portion of the latter and of the internal capsule. On the left side a dark mass was seen in the anterior end of the left caudate nucleus. Similar nodules were also seen in the caudate nucleus of the right hemisphere, and at the foot of the descending motor convolution in its gray layer. The interesting feature of the case was not only in the metastases but also in the localization of the latter in the gray matter alone.

SPASMODIC TIC OF THE MANDIBLE. DR. SAMUEL B. HADDEN.

A man, aged 29, from the dispensary service of Dr. Spiller, is unable to close his mouth properly, with a resulting inability to masticate and talk properly. This condition has been present for about three years and developed overnight.

At the present time, as the patient talks or chews, the mandible gradually becomes depressed. Support of the mandible by pressure beneath the chin or by pressure on the condyle, upward and forward, enables him to overcome the difficulty. This suggests a myasthenoid state. No history of psychic trauma or of encephalitis could be obtained. The electrical reactions are normal. Neurologic examination gives negative results. As the patient opens his mouth voluntarily,

about midway, there is a click. This is possibly a subluxation with dislocation of the intra-articular disk. A roentgenogram of the jaw joint shows it to be normal.

It is possible that the condition is an isolated symptom of encephalitis. Future developments may prove the correctness of this view. There is also, in all probability, a psychic element in the case; however, rather than a pure psychoneurotic tic we look on it as one superimposed on a subluxation of the jaw.

DISCUSSION

DR. N. W. WINKELMAN: This man was my patient several years ago, but his symptoms then were different. He then complained of an inability to talk, because the lower jaw would drop and the tongue would follow the lower jaw. On examination, there were no signs of an organic lesion. I learned that he was working for his father as a waiter in their small restaurant. He felt that this was much beneath his dignity. He wanted to be a professional baseball pitcher, but was unsuccessful in this calling. He admitted that his difficulty in talking was most manifest when he had to call out the orders for food in a loud voice. Strangely enough he could talk very glibly on baseball matters or when he took time off from the restaurant. He has been to many physicians. At the Philadelphia General Hospital a marked improvement occurred, but apparently he lapsed into his old complaints when he returned home.

DR. A. M. ORNSTEEN: I saw this man in 1927 and considered that his condition was functional. I remarked to his physician that this diagnosis should be made with reservation, as he might show later more evidence of chronic epidemic encephalitis. The wider one's experience with encephalitis, the more careful is one in making diagnosis of hysteria. In this case I still reserve a place in my mind for an organic basis of the jaw spasm. I have under observation two patients with chronic parkinsonism, both adolescent men, in whom there is involuntary opening of the mouth.

DR. ALFRED GORDON: Information obtained from one of the previous speakers is valuable from a diagnostic standpoint. The patient was employed in a place where his duties consisted of calling out continuously names and objects. The occupation was extremely distasteful to him and he wished to give it up, but could not for financial reasons. According to Dr. Hadden, the patient's lower jaw drops while he is talking. The underlying psychic factor is evident. The lowering of the jaw seems to be a defense reaction. The patient does not want to talk, yet he is compelled to do so. His employment being distasteful to him makes him unconsciously open his mouth and cut off the conversation by lowering the jaw. This psychic component appears to me to be the chief factor in the patient's abnormal behavior.

A CASE OF TABES WITH HORNER'S SYNDROME. DR. BERNARD I. COMROE.

A careful search through the *Index Medicus* fails to reveal a similar report of the presence of a Horner's syndrome in a patient with tabes.

The patient is a white man, married, of Ukrainian nationality, who, seven years before presentation, noticed pains under the left costal margin; sometimes these were burning in character and sometimes merely a sensation of flies crawling over him or of mice running on him. It was not a sharp shooting pain. To relieve the pain an appendectomy was done at the Philadelphia Hospital. The pains persisted, however, and eight months before presentation, at another hospital, a cholecystectomy was done to relieve the pain. This, however, was similarly unsuccessful. He is now under Dr. Spiller's care on the neurologic ward at the University Hospital. He complains of pains on the left side of the abdomen under the rib margin, together with queer sensations on the ulnar side of the right forearm.

Physical examination revealed: diminution of the sensations of heat, cold and pain on the ulnar side of the right forearm and to a very slight extent, on the

left; a loss of biceps, triceps, patellar and achilles tendon reflexes; a slight Romberg sign; a Horner's syndrome on the right (enophthalmos, narrowed palpebral fissure and miosis); a palpable spleen, slightly enlarged, which is very tender, probably associated with a perisplenitis.

Laboratory examinations revealed: By roentgenogram, bilateral adhesions of the diaphragm to the thoracic wall on both sides, and an appearance of bronchiectasis; the possibility of an old fracture of the body of the seventh cervical vertebra, though this is uncertain; cranium and pituitary region normal. The blood and urine were normal. The Wassermann test of the blood was negative with all antigens. Cerebrospinal fluid showed: 1.7 units of protein, 4 lymphocytes per cubic millimeter, and the Wassermann and colloidal gold tests to be negative.

We were puzzled at first by the repeatedly negative Wassermann tests of the blood and negative spinal fluid results. However, we learned later that the man had received treatment for syphilis in 1927, at which time the Wassermann test of the blood and the spinal fluid tests were strongly positive for syphilis.

The undoubted explanation of the Horner's syndrome is an involvement of the posterior roots of the seventh or eighth cervical or of the first thoracic segments, with destruction of the sympathetic fibers which leave there.

DISCUSSION

DR WILLIAM G. SPILLER: This man is presented not merely because he has a sympathetic ocular paralysis of the right side, but also because he has tabes with paresthesia on the ulnar side of each upper limb, more pronounced on the right side, in the distribution of the eighth cervical and first thoracic roots. The ocular sympathetic takes origin also from these roots, and while paresthesia is not uncommon in this distribution in tabes, it is seldom associated with paralysis of the ocular sympathetic supply. The roentgenologic examination shows no distinct lesion at the seventh cervical and first thoracic vertebrae.

A BROWN-SÉQUARD SYNDROME CAUSED BY A STAB WOUND. DR. LEWIS K. HOBERMAN.

This patient was carried into the Temple University Hospital accident ward on a stretcher by the police. He gave a history of having been stabbed in the back during a fight. After local treatment of the wound, he was admitted to the service of Drs. Fay and Winkelman for study and treatment. It was learned that consciousness had been retained throughout. He complained of loss of function of the left lower extremity and a feeling of numbness and heaviness of the right lower extremity. There was no hemoptysis or pain on deep respiration.

On physical examination, the head, neck, lungs and heart were found free from any gross pathologic process. There was an open wound on the left side of the chest posteriorly, the mouth of which was at the base of the spinous process of the left scapula. The direction of the wound was inward and downward toward the spinous process of the fifth dorsal vertebra. Motor power was lost completely, as were the abdominal and cremasteric reflexes on the left side. The knee and ankle jerks were not obtained on this side. A Babinski sign and ankle clonus were absent. On the right side, the abdominal and cremasteric reflexes, ankle jerk and knee jerk were obtained, but no Babinski sign or ankle clonus was elicited.

The sensory examination revealed an area of hyperesthesia at the level of the nipples, encircling the chest on both sides. The upper border of the fifth rib was the lowest level at which cold could be distinguished. A line bisecting the region between the xiphoid and umbilicus and traced posteriorly on the right side to the fifth thoracic vertebra was the lowest limit where heat could be appreciated. A line drawn from the umbilicus posteriorly to the fifth thoracic vertebra on the left was the lowest limit at which pain could be felt. Beneath these points pain and temperature sensations could not be elicited. However, there was preservation of muscle, position, vibratory and tactile senses. On the left side there was no

disturbance in sensation, including vibratory and muscle senses. The lesion was localized at the fifth dorsal segment.

The pilomotor phenomenon was brought out on the left side, the lowest level being the upper border of the fifth rib.

There was an area of pain sensitiveness in the left axilla and the left side of the chest posteriorly.

Treatment consisted of: (1) antiseptic care of the wound; (2) the administration of tetanus antitoxin—1,500 units intramuscularly; (3) lumbar punctures repeated every eight hours for the first two days, every twelve hours on the third and fourth days and once a day during the fifth and sixth days. Each specimen of spinal fluid was sent to the laboratory for a red blood cell count and smears were made for bacteria. No bacteria were ever found and the red blood cell count varied from 130,000 at the initial puncture to 10 cells in the final puncture. The pressure by manometric readings never rose above 8 mm. of mercury in the horizontal position. In addition to lumbar puncture, methenamine was given and the patient was treated symptomatically.

After the second lumbar puncture, the patient could move the toes and foot of the paralyzed limb, the left, and had a tingling feeling in the right lower limb. Motor power has returned gradually to the left leg as has sensation to the right leg, but this is impaired as compared to that in the left. Motor power has returned sufficiently in the left leg to enable the patient to bear his weight without difficulty.

One month after admission, on March 23, 1930, an examination revealed that the knee and ankle jerks were exaggerated, and patellar and ankle clonus as well as a Babinski sign were present on both sides.

Yaskin reported a similar case in 1923 of an atypical Brown-Séquard syndrome caused by a darning needle thrust into the lateral column.

This case is presented because it is an atypical Brown-Séquard syndrome with recovery; because it was caused by a dirty knife with no subsequent meningeal infection, and because, although excellent recovery has taken place, there still remains an atypical Brown-Séquard syndrome.

DISCUSSION

DR N. W. WINKELMAN: Dr. Fay tells me that it takes him about an hour and a half to perform an operation such as this under the strictest aseptic conditions. Yet here the operation was done in two or three seconds with a dirty knife with excellent results. There is no involvement of the posterior column as far as we can determine. The knife must have been extremely sharp and have cut only the lateral portion of the spinal cord. The result has been a modified Spiller operation with the addition of mild residual pyramidal tract signs.

DR. FREDERIC H. LEAVITT: Some years ago, I exhibited a patient before this society who presented physical signs similar to those of Dr. Hoberman's patient. The man was a physician, who reported a gradual onset of the symptoms. A roentgenogram revealed a knife blade, 3 inches long, "in situ" at the level of the fourth dorsal vertebra, ¼ inch to the right of the midline, thus penetrating the spinal canal. The patient was then 60 years of age and remembered that forty-one years before he had been stabbed in the back during a drunken brawl; he thought that the knife had been withdrawn after the stab. The wound healed promptly without infection and the incident passed from his memory until it was recalled by the roentgenogram evidence. No symptoms of a spinal condition had developed until thirty-five years later, when symptoms of a spastic paraplegia began to be manifest. Serologic study of the blood and spinal fluid gave absolutely normal results and the surgical opinion expressed was that any attempt to remove the knife blade in such a perilous situation, after all these years, would prove disastrous to the patient.

CHROMATOPHOROMA OF THE SKIN AND LEPTOMENINGES. DR. JOHN EIMAN.

This paper will be published in full in a later issue of the ARCHIVES.

LABYRINTHINE VERTIGO; AN UNUSUAL CAUSE. DR. FREDERIC H. LEAVITT.

This patient is presented because he shows an unusual cause of labyrinthine vertigo and deafness. The patient is a man aged 60, who has occupied a position of great prominence in the architectural world. He gave a history of having suffered with decreasing acuity of hearing for two years and for the past few months of severe vertigo of the labyrinthine type, which was made worse when the plane of his body was changed. He also noticed an increase in the size of his head as shown by the fact that it was getting too large for his hats. He had had four spontaneous fractures of the right humerus and a roentgen examination revealed a bony condition similar to Paget's disease. Further roentgen examination of the skull showed a complete destruction of the petrous bone on the left side and an incomplete destruction of the petrous bone on the right side. It is my opinion that this patient's cochlear and vestibular symptoms are due to destruction of these organs by the invading bone process. Roentgen examination of the spine and of the bones of the legs revealed similar conditions present in those parts.

The general attitude, posture, facial expression and movements were suggestive of Parkinson's disease. The patient was totally deaf in the left and moderately deaf in the right ear. He complained of no heachache, vomiting, dimness of vision, diplopia, visual field cutting or other evidence of increased intracranial pressure. Station with the feet together showed a very pronounced Romberg sway, nearly always a falling backward and unsteadiness. Any movement of the head from one plane to another produced an increase in the vestibular condition. He stated that he had no tinnitus whatever. Coordination of the right arm was difficult to determine because of repeated fractures of the right humerus, producing an ankylosis near the shoulder. There was no ataxia in the finger-to-finger and finger-to-nose tests in either arm, nor in the heel-to-knee test in either leg. Diadokokinesia showed no abnormalities. The grip was good and about equal in the two hands. There was a very marked osseous overgrowth of the fracture of the entire right upper arm with an ankylosis at the right shoulder joint. The arm jerks could not be obtained on either side. The knee jerks were active and equal. Stroking the sole of either foot produced plantar flexion of all toes. There was no ankle clonus on either side. In bed there was good motor power in all extremities, except at the right shoulder joint. The abdominal reflexes were present. The right pectoral muscle jerk was smaller than the left. The pupils were equal, regular in outline and reacted promptly to light and in accommodation. Extra-ocular movements were normal. There was no hemianopia in either eye. There was no nystagmus and no von Graefe's sign. There was a very persistent piston tremor of the tongue and some disturbance of articulation. The tongue protruded in the midline and moved well in all directions. The soft palate moved well. The teeth showed considerable crowning and bridging and were in a very filthy condition. The tonsils were small and showed no evidence of disease. Vision was fair in both eyes. The sense of smell was normal. The third, fourth, sixth, seventh and ninth cranial nerves functioned normally. The eighth nerve showed impaired function. The tenth, eleventh and twelfth cranial nerves apparently functioned properly. The thyroid was not enlarged. Heart action was regular; no murmurs were audible; the rate at rest was 74 with no undue increase of rate on effort; rhythm was myocardial and the sounds lacked tone. The blood pressure was 130 systolic and 90 diastolic. The lungs were normal grossly. The abdomen showed no masses or areas of tenderness. There was a very marked limitation of movement of the neck so that the head was held rigidly and rotation or canting of the head was very poorly accomplished; there seemed to be a decided stiffness of the entire spine. The gait was decidedly of a vestibular type of ataxia, which was markedly increased when the eyes were closed.

On Jan. 19, 1929, a roentgen examination of the right humerus showed a marked degree of scattered areas of rarefaction with cortical thickening and trabeculation, evidently due to a localized Paget's disease. No lines of old fracture could be seen. There was outward bowing. The skull showed large areas of rarefaction with an area of normal density of the vertex or possibly a slightly increased density in the vertex. This area had sharply defined areas. There were also small dense areas scattered through the skull. I believe that this is a somewhat typical Paget's disease and suspect that it may be undergoing sarcomatous degeneration. There was marked destruction of the petrous bone on the left side and to a lesser degree on the right.

An examination of the ears on Jan. 18, 1929, revealed: both ear drums normal; 20 double vibrations not heard in the left ear; the Weber test plus to the right ear and bone conduction greater than air conduction in this ear. There was no spontaneous nystagmus, but on looking forcibly upward the right eye deviated outward and he saw double. Spontaneous past pointing with the left hand occurred 4 inches to the right. Questionable past pointing with the right hand occurred to the right. Douching the left ear caused past pointing 3 inches to the left. There was no nystagmus, vertigo or change in past pointing.

Douching the right ear for forty seconds caused slight horizontal nystagmus in the supine position. There was no change in nystagmus, vertigo or past pointing. The blood count was normal in all respects. Urinalysis showed no Benz-Jones albumose.

DISCUSSION

DR. EUGENE P. PENDERGRASS: It is easy to demonstrate the loss of osseous tissue in the parietal region of the skull as well as the increased density of the bones of the base, particularly in the mastoid region. Possibly this may have something to do with the patient's symptoms. In addition, other bones are involved. In the right humerus there is an irregular rarefaction of the medullary canal and some periosteal proliferation, causing an increased thickness of the cortex. The process also involves the scapula. This is a peculiar type of lesion, and one has to consider the fibrosing type of osteitis and Paget's disease. Dr. Pancoast and I believe that this is probably a type of fibrosing osteitis. Xanthomatosis must be excluded, but this condition is usually associated with diabetes insipidus. The lesion does not look like a syphilitic osteomyelitis or a metastatic lesion. The changes in the skull in the region of the semicircular canals probably account for the patient's symptoms.

TWO CLINICAL CASES OF MULTIPLE PSEUDOFRACTURES OF BONES. DR. WILLIAMS B. CADWALADER.

My object in reporting the following two cases is to call attention to a disease of the bones of obscure origin regarding which little seems to be known. Apparently the disease begins in early life and is slowly progressive in course, affecting many of the bones of the skeleton, and cannot be demonstrated by roentgen examination until it has reached a certain stage in development. Clinically, it is characterized by tenderness over the affected bones and pain which is aggravated by movement, particularly when a movement is performed quickly, and is associated with exaggeration of the tendon reflexes without any marked degree of muscular atrophy and without additional symptoms.

CASE 1.—Grace M., aged 38, when 13, immediately after being struck in the sacral region, was said to have lost completely the use of the legs, but recovered after an hour had elapsed. When 32, she complained of weakness and dull pain in the lumbar region, which was aggravated by flexion or extension of the spinal column. Soon after this she noticed that while walking there was a tendency to sway from side to side. Later, tenderness developed over the tenth, eleventh and twelfth ribs of both sides. A year later, on arising from a sitting posture and attempting to walk, it was impossible for her to flex the left leg and she experienced a sudden sharp pain in the region of the ilium, which radiated through

the lower limbs. If she forced herself to take the first step quickly after standing up, she frequently fell to the floor. If, after rising from the sitting position, she remained standing still for three or four minutes, she could slowly begin to walk without having much pain, but even then it was difficult to move the limbs for the first few steps. Gradually, however, the ability to walk became easier and with little or no pain. Any quick movements of the legs caused pain; slow movement, however, did not cause pain. Any movement in abduction of the legs caused pain in the hip joints. These difficulties gradually increased. Movements of the legs against resistance caused pain in the thighs.

Objective signs were not found, except a moderate increase of the patellar reflexes and tenderness over the long bones. The electrical reactions of the muscles were normal, and there were no atrophy of the muscles, paresthesias, or muscular paralysis. Because of the difficulties in initiating movements, the condition was regarded as a possible case of myotonic dystrophy. The diagnosis of bone disease was not made until some weeks subsequently by Dr. Pancoast and Dr. Pendergrass after numerous roentgen examinations of the bones.

CASE 2.—Estelle M., age 35, was said to have been well during infancy, but the muscles of the legs below the knees were never well developed, and there was a tendency to genu valgum. When 14, she complained of subacute pain in both hips and in the spinal column. At that time she was thoroughly studied, but no definite diagnosis was made. The pain continued off and on and was never very severe, but was always aggravated by movements. In spite of this she was able to lead a fairly normal life until 1928, when the pain had become so severe in the left hip that she was admitted to the Hospital of the University of Pennsylvania. The condition was attributed to a gonococcus infection, but the correct diagnosis was not made until the time of presentation. The symptoms gradually increased in severity, and since August, 1929, the patient has not been able to walk without assistance, not, however, because of muscular paralysis, but apparently because of pain.

In January, 1930, the patient again came under my observation. The examination then revealed moderate uniform atrophy of the muscles below the knees of each leg. This had the appearance of the atrophy of the Charcot-Marie-Tooth type. The muscles of the thigh were well developed. There was moderate genu valgum. The achilles, patellar, biceps and triceps tendon reflexes were all equally and moderately exaggerated, and there was tenderness of the spine and over the ribs, femurs and pelvis, and pain on movement of the limbs, particularly when abducting the legs. Because of these facts, the possibility of a disease of the bone was suspected; this was confirmed by the roentgen examinations. All laboratory examinations gave negative results, including tests of the calcium metabolism, yet the blood count showed 48 per cent of mononuclear leukocytes.

It is uncertain in my opinion how much importance to attribute to what appeared to be lack of development of the muscles below the knees, since it probably had existed since childhood.

DISCUSSION

DR. EUGENE P. PENDERGRASS: We had the opportunity of studying both of the patients reported by Dr. Cadwalader. They were then on the service of Dr. William G. Spiller in the University of Pennsylvania Hospital. In both cases the request for the roentgen examination was for a study of the sacroiliac joints, and unfortunately we missed the pathologic process in both cases.

The patients complained of a great deal of pain in the back and this was attributed to weakness of the muscle and spasm of the ileopsoas muscle. Subsequently, the patients were studied elsewhere, one by Dr. Milkman in Scranton, Pa., and the other by Dr. Newcomet at the Presbyterian Hospital of Philadelphia.

The earliest roentgen manifestations of the disease are small punched-out areas that are usually bilateral and symmetrical in the long bones. In the cases that we have studied, the most frequent sites of the disease have been the ribs, femurs, radii, ulnae and fibulae, but any bone may be affected. The small punched-out

areas gradually increase in size until the cortex is entirely destroyed, giving the appearance of an irregular rarefied transverse zone with complete loss of bony substance. This has been called a pseudofracture.

Following the World War, a number of articles appeared in the German and Austrian literature describing conditions that were thought to be due to late rickets, osteomalacia or, as recently termed by Looser, multiple symmetrical pseudofractures. The condition was thought to occur in elderly, poorly nourished persons. It occurred in women after the menopause and occasionally in young men. The condition has to be differentiated from fragilitas ossium, osteogenesis imperfecta, senile osteoporosis, osteo-opso-thyrosa and late rickets.

A number of authors believe that rickets and osteomalacia are one and the same condition. The symptom-complex consists of lumbosacral pain, general weakness and a waddle gait. The patient has difficulty in raising the legs. There is tenderness over the lesions of the bone. There are increased reflexes. The etiology is supposed to be a lack of food or possibly a disturbance in endocrine function or lack of vitamins. The increased reflexes have been explained on the basis of a disturbance of parathyroid secretion. Usually there is some hysteria which has been explained by some authors as being a defense mechanism.

The condition is not common in this country and is probably related to osteomalacia or possibly some parathyroid deficiency. Osteogenesis imperfecta and fragilitas ossium are usually familial; these patients have blue sclerotics. In fragilitas ossium there is usually difficulty in hearing, and new bone formation occurs in the repair of the fractures. In the condition of pseudofractures there is no bone repair. Idiopathic osteo-opsa-thyrosa usually occurs between the ages of 12 and 20 in males. Pseudofractures can be differentiated from senile osteoporosis by the fact that there is no generalized porosis of the bones.

This condition is most unusual, and further study will have to be made before the cause can be demonstrated. Neither of these patients was suffering from lack of food; they were of the intelligent type; there was no family history of the condition, and they have not responded to any form of treatment, which consisted of calcium therapy, parathyroid therapy, ultraviolet rays, vitamins, etc.

Spasmodic Tic of the Ear. Dr. Lewis H. Hitzrot.

The case to be presented is one of irregular involuntary contraction of the elevator muscle of the left auricula. The patient, who first came under my observation on February 28, is an Italian woman, aged 39, whose presenting complaints were weakness and nervousness, transient precordial pain and a sensation of burning of the scalp above the left ear. The first group of symptoms may be ascribed to neurosis; associated with the last named, the paresthesia of the scalp, however, she has had for about six months a spasmodic, uncontrollable elevation of the left ear. It has been learned definitely that it does not go on during sleep, but begins immediately on waking.

The past medical history reveals a discharging left ear in childhood. There were no severe illnesses. The patient's neurotic tendencies have been accentuated during six years of an unhappy married life. She has been fearful since learning some years ago that her husband had had syphilis. No history of a change in the state of health preceding the onset of the tic could be elicited. The patient is not subject to respiratory, throat or ear infections. She had no period of somnolence. She does maintain that she is mentally duller than heretofore.

Examination showed a moderately obese woman, depressed and somewhat anxious, whose facial expression could be described as dull, but hardly fixed. There were no stigmas of syphilis; likewise there was no true focus of infection, although a nose and throat consultant considered that the tonsils were probably infected. The scalp was clean. The physical examination otherwise gave entirely negative results, except for a small ovarian cyst and a lacerated cervix uteri.

Both auditory canals were normal; the tympanic membranes were slightly retracted. At irregular intervals of a few seconds, the contractions often occurring in groups, the left external ear was pulled vertically upward through a distance

of 1 cm. No other movement, either in the intrinsic or the extrinsic muscles of the ear, could be demonstrated. The patient recently has complained of a similar paresthesia (sense of heat) above the right ear, but no movements have been seen. The Wassermann reaction of the blood was negative on two occasions and other laboratory data offered nothing of significance.

This is evidently a nerve irritation affecting a small branch of the left facial nerve, the temporal, which supplies two vestigial muscle bands, the auricularis anterior and superior. The condition is localized even within the fibers of that temporal branch, for the auricularis anterior, the muscle frequently under voluntary control, is quiet. No source of local irritation was found which could stimulate muscle or nerve.

Dr. Ornsteen has studied the patient and has made moving pictures of her peculiarities which he will show. I hope also that he will give his observations bearing on encephalitis as the etiologic factor. Our failure to find a local irritative focus and the limitation of the spasm to a vestigial muscle bundle not under voluntary control give reasons not to ascribe the tic to focal infection or hysteria.

DISCUSSION

DR. A. M. ORNSTEEN: An isolated tic movement of a single muscle of the ear is most startling. It cannot be accepted as an hysterical manipulation without ruling out an organic possibility first. Ordinarily, a functional tic involves a muscle group which subserves an act having a definite functional or purposeful value or significance, such as blinking, swallowing, hiccoughing, belching, shoulder-shrugging, etc. Movement of the ear is phylogenetically ancient and as such is not ordinarily utilized to express an hysterical tension. The rhythmicity of the tic and its focal character suggest its striate origin. Since focal tics are commonly observed in epidemic types of encephalitis we looked for early signs of parkinsonism. These were found in the form of asymmetric finger postures while the hands are in repose by the side and while outstretched; also a limitation of rapid thumb-forefinger movements on the right, constituting the characteristic triad of incipient parkinsonism. In harmony with this observation on the right was the recognition of an innervation defect on the right side of the mouth. She occasionally had frequent blinking movements. The facies impressed one as lacking in normal subtlety of emotional expression.

THE EFFECT OF STRAMONIUM IN PARKINSONISM. DR. EDWARD D. HOEDEMAKER and DR. M. A. BURNS.

To Dr. Stoerck (A Manual of Pharmocology, ed. 3, p. 373) we are indebted for the introduction of datura stramonium in the treatment of epilepsy, mania and some other convulsive affections. In 1797, Cooper (A Dissertation on the Properties and Effects of the Datura Stramonium and on Its Use in Medicine, p. 39) employed the drug at the Pennsylvania Hospital, Philadelphia, for the relief of mania, epilepsy and certain fevers. Since then, stramonium has been used particularly for those ailments and also for headache, insomnia and muscular rheumatism.

In recent years, stramonium has been employed in the treatment of parkinsonism, especially of the postencephalitic type. In 1925, Juster (*Rev. neurol.* **1**:218, 1925) reported results with stramonium and its effect on parkinsonian rigidity. He used datura stramonium, recommending the drug as the "daily bread" of parkinsonians. The next year, Laignel-Lavastine and Valence (*Bull. et mém. Soc. méd. d. hôp. de Paris* **50**:921, 1926) reported their observations with datura stramonium, in the same form as used by Juster. They noticed diminution or abolishment of rigidity and tremor, bradykinesia and excessive salivation, and were impressed with the improved mental attitude exhibited by their patients. Harris (*Lancet* **1**:698, 1927), however, when using the tincture of stramonium, found the results disappointing. As he prescribed only 25 minims (1.5 cc.) thrice daily, it is possible that the dose was not large enough to act effectively. In 1928, Carmichael and Green (*Quart.*

J. Med. **85**:51, 1928) reviewed their work with the tincture of stramonium and found that in large doses, usually between 45 and 60 minims (2.8 and 3.8 cc.), three times daily, the drug lessened the rigidity, increased the ability to perform fine movements and improved the mental condition of the patient. They found also that tremor was not affected, and that toxic symptoms were rarely severe and could be overcome in various ways. Shapiro (*J. Nerv. & Ment. Dis.* **68**:488, 1928), in 1928, believed that datura stramonium was the most effective palliative remedy in parkinsonian states, and claimed that all the symptoms were improved simultaneously. The tincture of stramonium was employed by Jacobsen and Epplen (*J. A. M. A.* **93**:2027 [Dec. 28] 1929); they believed it an excellent palliative measure for all symptoms of the parkinsonian syndrome of postencephalitic origin, with the single exception of the pareses which they thought remained unbenefited. In 1929, Moren (*Kentucky M. J.* **27**:291, 1929) reported results with datura stramonium and stated that the main improvements were in muscular rigidity, salivation, posture, speech and mental condition.

Just why stramonium should be so effective in parkinsonism is still obscure. One reference (*Epitome of the U. S. Pharmacopeia & National Formulary, 1926, p. 171*) states that the tincture of stramonium consists of about 0.025 per cent of stramonium alkaloids, the absolute alcoholic content being about 45 per cent, and further study reveals that its actions and uses are similar to those of belladonna, over which it has no advantages. Sajous (Internal Secretions and Principles of Medicine, vol. 2, p. 1215) said that the physiologic action of stramonium is like to that of belladonna, but its effects are less marked, its stimulating influence being less violent. On consulting other authorities one learns that daturin, an alkaloid of datura stramonium, acts like atropine, but is stronger and is said to be a mixture of hyoscyamine hydrobromide and atropine. Its chief alkaloid is hyoscyamine hydrobromide, but it also contains small traces of scopolamine hydrobromide and atroscine. Further study should be made in order to determine the true source of benefit obtained by the physiologic action of stramonium.

In reporting this series of cases we have limited ourselves strictly to the use of tincture of stramonium. We are not comparing it with any other drug used in the treatment of parkinsonism. The cases reported were not specially selected, but were those of patients suffering from either classic paralysis agitans or the parkinsonian syndrome following epidemic encephalitis, as they appeared in the wards of the Philadelphia General Hospital. All cases on all available wards were used.

The study of the series of thirty-six cases began on July 1, 1929. Three patients died from intercurrent diseases; two were discharged; consequently we are now reporting the effect of the tincture of stramonium in a series of thirty-one cases. Four were cases of classic paralysis agitans, and twenty-seven followed epidemic encephalitis. All patients were given the tincture of stramonium, 20 minims (1.25 cc.) thrice daily, each dose being increased 5 minims (0.3 cc.) a week until 60 minims or more were taken three times a day. Patients were observed weekly for any change which might occur in their general behavior, and especially for changes in rigidity, tremor, excessive salivation and ability to perform fine and rapid movements, and also for signs of toxic effects. The preparation used was made in the pharmacy of the Philadelphia General Hospital from datura stramonium leaves; a 10 per cent tincture was employed.

The four patients with classic paralysis agitans were of the following ages: 51, 55, 56 and 70. In these patients the results were poor. All four took as high as 60 minims of the tincture of stramonium three times a day. Those suffering from excessive salivation were relieved, and one felt slightly more relaxed. Except for these observations there was no improvement. The dose of the drug was not increased beyond 60 minims because of marked xerostomia and loss of appetite.

Of the twenty-seven patients with postencephalitic parkinsonism, nine were women, ranging in age from 20 to 43 years, and eighteen were men whose ages varied between 20 and 70 years. In general, there was no definite relation between the duration of the illness and the improvement following administration of the

drug, but patients revealing the most marked symptoms of the disease showed less response. We have classified these twenty-seven cases in four groups: (1) those in which the response was excellent; (2) those with good results; (3) those with only fair results, and (4) those with little or no effects.

The eleven patients in group 1 all gave excellent results, viz., marked diminution in rigidity throughout the entire body, return of associated movements to a surprising degree, complete relief from excessive salivation, and marked improvement in the general mental condition. In group 2, which consisted of five cases, the patients showed relief from excessive salivation, marked improvement in rigidity and some return of associated movements. All the patients felt more relaxed and capable of doing things for themselves which they could not do before the administration of the drug. In the five patients who made up group 3 the results were only fair. All showed complete relief from excessive salivation, and, due to the effect of the drug on rigidity, felt more comfortable generally, but they could not handle themselves as well as those in group 2. Group 4 consisted of six cases. These patients were all relieved from excessive salivation, but aside from that it was difficult to note any definite change that could be attributed to the drug.

THERAPEUTIC EFFECTS

Rigidity.—This includes posture, gait and the ability to perform rapid and fine movements. All patients showed some improvement in this symptom. In twelve cases the relief was striking. Four of these patients were completely bedridden, could not walk without falling, could not feed or dress themselves and could not turn over in bed alone. On being given high dosage of stramonium these patients were able to get out of bed without assistance, to walk easily without falling, to feed and dress themselves and to help about the ward. They were also able to perform rapid and fine movements, the execution of which had been impossible previously. One patient in particular, M. M., a woman, aged 28, who had been ill for five years and bedridden for at least a year, was able to sew and play a selection on the piano. She took as much as 90 minims (5.6 cc.) three times daily. Another interesting case was that of C. B., a man, aged 31, who had been ill for seven years. Now, he walks well without danger of falling and, aside from feeding and dressing himself without difficulty, has been able to return to work; he is employed in the capacity of night watchman. The maximum dose administered to him was 90 minims three times daily. Eight were patients who were able to walk with difficulty, but could not dress or feed themselves. The gait and posture were typically parkinsonian. Under treatment, these eight showed marked relief from rigidity; their posture became more erect, and in two or three instances appeared practically normal to the casual observer. All were able to perform rapid and fine movements and help about the wards, and two of them are now acting as messengers throughout the hospital.

Of the remaining fifteen cases all but six were relieved to such a degree that they were able to feed themselves; the remaining six showed no improvement whatever except relief from excessive salivation. All patients, with the exception of these six, remarked that they were much more comfortable, felt more relaxed and did not want the drug discontinued.

Tremor.—This includes tremor of all parts of the body—lids, tongue, lips, extremities and head. Tremor was definitely improved in three instances. One of these patients, D. E., a woman, aged 31, who had been ill for three years, had moderately intense tremor of both upper extremities and tongue before medication was begun. When she was taking 60 minims three times a day, the tremor was definitely lessened in the upper extremities, and according to the patient it was absent part of each day. With 90 minims, three times a day, all tremor was gone except in the tongue, and appeared only on marked exertion or excitement. Another patient, N. R., a woman, aged 37, who had been ill for three years, had a moderate tremor of the lips, lids and all extremities. With 90 minims, three times daily, all tremor with the exception of that of the tongue disappeared.

On marked effort there was slight return of the tremor. The third patient, P. H., a man, aged 34, who had been ill for six years, had a slight tremor of the left lower extremity and marked tremor of the left upper extremity. With 90 minims, three times a day, the tremor was completely gone in the left lower extremity and was moderately reduced in the left upper extremity, but was made worse on effort.

In the remaining twenty-four cases, little if any improvement in tremor was observed. However, many of the patients remarked that they did less shaking each day. We believe that this was due to the fact that they were more relaxed and at rest owing to the effect of the drug on rigidity.

Excessive Salivation.—Excessive salivation was completely relieved in all cases in which it existed before administration of the drug. Relief was noted almost from the onset with the use of small doses. The removal of this very troublesome symptom had a remarkable effect on the mental condition of each patient.

Automatic Associated Movements.—The improvement in this respect paralleled closely the improvement in rigidity and posture. In some instances it was difficult to detect casually any evidence of the typical parkinsonian syndrome when the patient was walking, in cases in which the symptom had been full fledged previously.

Oculogyric Crises.—Relief of oculogyric crises was noted in one patient, E. F., a woman, aged 27. This patient suffered from oculogyric crises every one or two days, the condition lasting from three to four hours. With 60 minims, thrice daily, the crises occurred only every seven or eight days and lasted but ten minutes. On discharge, she was without stramonium for a month, during which time the crises returned with their original severity; they were again relieved by the use of the drug.

Mental Effect.—The mental attitude was definitely improved in twenty-one cases. This effect paralleled directly the improvement in the physical condition of the patient. Those benefited were hopeful and more alert; they cooperated about the ward and became interested in the effect of the drug, and in noting what acts they could perform which had been impossible of execution before beginning medication; many of them began to plan their discharge from the hospital.

DOSAGE

The average dose for the whole group was 76 minims (4.68 cc.) three times daily; the average for men was 80 minims (5 cc.) and for women 72 minims (4.45 cc.) three times daily. The greatest dose administered continuously was 110 minims (6.76 cc.) and the smallest was 45 minims (2.8 cc.). In general, younger patients had greater tolerance than older ones, and men had greater tolerance than women.

TOXIC EFFECTS

Xerostomia.—While this effect was present practically from the start with small doses, we considered it toxic only when the symptom became so severe and troublesome that the patient preferred no increase in the dosage of the drug. Many patients counteracted this affect by holding candy (especially lemon drops) in their mouths a few times a day or by chewing gum to stimulate salivation. The symptom disappeared completely after the drug was withdrawn for twenty-four hours. No lasting effects were noted. Xerostomia was the cause of limiting the dose in eighteen cases. Tolerance was developed in all instances up to a certain point, at which time the symptom continued with about the same severity. It was noted that with each increasing dose the symptom became noticeable again, but abated after the dosage had been continued for a few days, and it was not a cause of discomfort.

Blurring of Vision.—This effect was observed after the dosage had reached a point near the maximum, and occurred later than xerostomia. In some cases the patient preferred the symptom rather than to go without medication or have the dosage reduced. There was no benefit from the use of glasses and no attempt

was made to use physostigmine salicylate for relief. The symptom disappeared in from fourteen to twenty-four hours after withdrawal of the drug. There were no lasting effects. It was the cause of limiting the dose in four cases. The blurring was of a fleeting nature, never lasting more than from one-half to one hour, and usually came on following each dose.

Dizziness.—This effect appeared only with high doses and then moderately, if at all. It was the reason for restricting the dose in but one case. It was present in five cases.

Loss of Appetite.—This symptom appeared only with high doses and in all but one case it disappeared after a day or two without other medication or without limiting the dose. It was the cause of restricting treatment in one case; it was present in six cases.

Nervousness and Insomnia.—This occurred in one patient and was the reason for limiting the dose. The patient described it as a restlessness.

Pruritus.—This appeared on the soles of the feet of one patient. There were no focal lesions, and it was relieved only by limiting the dose of stramonium.

Nausea and Vomiting.—There was no evidence of nausea or vomiting in the entire group of cases as a result of the drug.

SUMMARY

Of the twenty-seven patients with paralysis agitans following epidemic encephalitis three have been discharged and have returned to work. One woman went back to her home and has taken over all the duties of her household. The second, a man, is employed as a night watchman, and the third, a man, is working in some other capacity. All three return to the neuropsychiatric clinic for observation about once in three weeks. Of the remaining twenty-four, ten patients are in a condition to be discharged, and the others are still under observation.

CONCLUSIONS

Tincture of stramonium in large doses lessens the rigidity in parkinsonism and increases the ability to perform fine, rapid movements.

With the exception of a few cases, tremor is not noticeably affected by the use of stramonium.

The mental condition of the patient appears to be improved by the drug.

The maximum dose necessary to obtain the best results varies in individual cases from 60 to 90 minims three times daily.

Toxic effects with large doses of stramonium are rarely severe and may be combated in various ways, as already mentioned. They appear to be more common in cases in which the disease has been most severe.

Tincture of stramonium has practically no effect in true paralysis agitans.

There are cases of parkinsonism, even of the postencephalitic type, in which stramonium will have no effect.

Tincture of stramonium, while only a palliative and not a panacea for parkinsonism, is an important drug for patients of this type, and should be used more extensively.

THE COORDINATION OF ANTAGONISTIC MUSCLES. DR. GRAYSON P. MCCOUCH.

The purpose of the paper is to indicate the nature and scope of studies on reciprocal innervation. Salient features of Sherrington's analysis of the flexion reflex are cited to illustrate the divergence of functional pattern from that suggested by anatomic nomenclature. Patterns involving the fixation of joints are contrasted with those mediating movements. Modifications of reflex patterns by proprioceptive components are considered briefly in situations where the normal attachments of muscles are preserved. The results presented are: preliminary experiments used here merely to indicate some of the problems awaiting solution.

Book Reviews

PSYCHOLOGIE DER SCHIZOPHRENIE. By JOSEF BERZE and HANS W. GRUHLE. Monographie aus dem Gesamtgebiete der Neurologie und Psychiatrie. No. 55. By O. Foerster and K. Wilmanns. Paper. Price, 16 marks. Pp. 168, with 11 illustrations. Berlin: Julius Springer, 1929.

The two leading speakers in the discussion at the Vienna meeting of the German Psychiatric Association (Sept. 13, 1927) in this book present in detail their reports to distinguish between the process symptoms and the defect symptoms in the mental symptomatology of schizophrenia. It would seem desirable to summarize the reasoning and the presentation of facts of Berze and Gruhle, as good examples of the wrestling with probably the most interesting and far-reaching problem of psychiatry.

Berze aims to make groups of cases, and of phases in each case. He distinguishes the concepts of psychotic fundamental disorder ("phenomenologically not demonstrable"), the primary symptoms (the immediate psychic expressions of an organic process not continually obvious) and the secondary symptoms (secondary adaptations that really furnish the general picture). Add to this the distinction of schizoid and schizophrenic reaction types and one sees what a tangle of "basic" concepts he starts out with.

From a "psychopathic-genetic" angle, Berze specifies: (1) active process schizophrenias, acute or slow, continuous, intermittent or remittent; (2) inactive "process schizophrenias" in a preliminary or final arrest (including the majority of Kraepelin's paraphrenias); (3) "reactive schizophrenias" without active organic but merely psychogenic developments; (4) complex types.

Berze's discussion is divided into the following chapters: I. Grouping of schizophrenias according to essential psychologic differences (yielding the four types of problems already outlined). II. The schizophrenic primary symptoms as an expression of the schizophrenic process (impressing one as a very meager development from Bleuler's plan and ambitions). III. Delimitation of the schizophrenic primary symptoms (a most nongratifying effort). IV. The schizophrenic thinking disorder. V. Differences of the thinking disorder in schizophrenic process phases and in schizophrenic defect states. VI. Psychologic theory of schizophrenia.

Berze justly emphasizes the fact that in a large percentage of schizophrenias one cannot find a trace of real primary symptoms, i. e., of the "process." He defines as psychotic primary symptoms those immediately obvious symptoms "which can in no way be further reduced psychologically." The psychotic fundamental disorder is something not directly "phenomenologically" demonstrable, but obtained only by inference. It should not be expected to be demonstrable as a unique and specific cardinal symptom.

The reviewer has not for a long time been confronted with a piece of work that shows as clearly as this "psychology of schizophrenia" the consequences of arbitrary disregard of a practical approach in dealing with human facts, as soon as structural, structural-functional and personality-functional data have to be given their due consideration. Berze abstains almost altogether from reference to concrete objective data and presents a painstaking discussion of the expressions of opinion of other noteworthy exponents, with an urge to reach some verbal abstractions suitable for the occasion of having to discuss schizophrenia. Gruhle, on the other hand, starts in the main from actual fragments of records, but he is so tied up with what one should and should not consider according to the philosophizing rules of his intellectual environment that one feels oneself painfully limited and preoccupied by arbitrary categories of "phenomenalism" and the

characteristic separation of understanding, empathic and causal types of dealing with the facts before one. One gets the feeling that a definite logic of science becomes more important than interest in the facts. There is a tremendous contrast between the simple and direct American instrumentalism of Charles Peirce, John Dewey and William James on the one hand, and Husserl and Jaspers and the formal psychologizers of the elementalist and the Dilthey type, and the classic and the freudian psychopathologists.

Gruhle, who uses at least fragments from cases of the Heidelberg clinic, considers schizophrenia from the outset as an organic disease, a reaction to an exogenic noxa or more probably an autochthonous encephalogenic disorder, in contrast to those who see in it a conflict psychosis, the flight of the personality from the conflict between an instinct and the censorship in the unconscious into "the symbol of the psychosis." He is definitely set on keeping the question of mind and body, or mind and brain, out of the problems of psychology proper—so as to be able to assume a specially dogmatic type? He is one of those who say "there is a psychology also of dementia paralytica." Hence there is "a psychology of schizophrenia," "provided that schizophrenia is a unit." Can such a psychology be causal? Is it just symptomatologic? What does psychology mean to these writers?

For Gruhle, the goal is that of turning from "the content" to the "pure" description of the abnormal functions, and he discusses as irreducible topics: (1) hallucinations; (2) the schizophrenic basic mood (the ego derangement); (3) the impulses; (4) the thinking disorder and language; (5) delusions; (6) theory of schizophrenia; (7) problem of the individual course; (8) psychology of the expression in schizophrenia.

The relatively greater concreteness makes Gruhle's share of the monograph more valuable, or at least potentially promising.

Gruhle's discussion of "genuine hallucinations" raises at once the question whether the samples he gives do not come from patients in whom the hallucinations mentioned are but part of a very far-reaching disorganization, so that one would not be tempted to use the introspective subjective comment of such a patient offhand for very far-reaching psychologizing hypotheses.

In such a complex of facts as that presented by the schizophrenic person, one must admit frankly that he is strongly dependent on a total impression and total reaction in a consideration and evaluation of the specific part-facts. In the selection of topics of preoccupation, it would seem eminently important that one should choose the items with which one can do something, and especially those which do something among themselves, i. e., speak through objective effects and results not in need of much interpretation.

Gruhle takes up the self and its disorder, especially its loss of spontaneity and mastership, its warmth and contact. The "basic mood" of the schizophrenic is not intelligible, not the result of more or less definite life-conflicts, not derived from complexes or from the structure of the personality. The wealth of examples given in the literature, especially the recent discussion of automatism by the French and in Gruhle, is illuminating, but should not be taken hastily to prove that one is dealing with something wholly foreign to what the normal person can feel or the "accountable" reactions show. One might say that: "Such is the nature of subject-organization and the range of its vicissitudes," before one claims that "this must be explainable only through passing outside of what we know of the psychobiologic working." By limiting psychology as Gruhle does, he may necessarily cut out the facts that others use for their genetic-dynamic formulation of psychobiology and psychopathology.

The impulsiveness and poverty of impulses, like the hallucinations, the fusions and the lack of fusion—one might say the disorders of presentative, of executive and of cogitative activity—are apt to be abrupt, poorly connected and poorly intelligible. One is here confronted with the perplexities of causality in the psychobiologic organism and life-cycle. It is humanly difficult to rise from the all-or-none neurophysiology to the relativities of man's total-function, a function

of a constantly variable unit or representation system. "To be all there" is a highly variable status. Usually we are but "partly there," and sometimes parts of the nonmentally integrated life of the organism rise and play the rôle assigned to the proverbial mince-pie, but also require consideration in the accidents of circulatory and glandular processes. We are not disembodied or independent souls and masters, but products of integration with varying consistency and dependability. There are different sets of causality, and it would seem unwise to force the right to symbolic conception or the consideration of the urge to power into an obligatory, mandatory position for all psychobiologic happenings. There are, no doubt, more psychogenic influences and inhibitions than were recognized before Freud; there are many drives and counter-drives or tendencies and counter-tendencies that deserve to be treated like those concerning which we have no hesitation to use our common-sense approach, although we have not the same personal or subjective clearness. But we also have the principle of fatigue, of "queering" through circulatory accidents, toxic influences, structural modifications and all the points we cover by our integration concept.

Gruhle claims that ambivalence has nothing in common with inconclusiveness, indecision, vacillation, doubt, etc., and he declines Bleuler's more compromising attempts at formulating this type of fact. The contrast between "Sperrung" (a sudden stop) and inhibition (applying a more protractedly working brake) is logically much more abrupt than in its actuality. Gruhle obtained the best subjective description of inhibition in a patient who later presented a completely dilapidated schizophrenia. The qualitative disorder of impulse, "Sperrung" or blocking, appears to be a specific fundamental symptom of schizophrenia, while excitement and stupor may be fundamental symptoms but not pathognomonic for schizophrenia. "There is such a thing as blocking without ambivalence but no ambivalence without blocking"—too much assurance derived from epigrammatic verbalism.

With the consideration of the thinking disorder, Gruhle comes to touch a point that leads to the comparison of his own views with the organicist effort of Kleist to trace the language changes in schizophrenia to what is known in aphasia.

Those who hold to a specific sphere of "thought" evidently treat language largely as a "means of expression," whereas it would seem that thought and language are inseparably interdependent and are best treated as a reaction or activity that occurs as full-fledged or silent or even pictorial, truly meant or only quoted or repeated. That it has its receptor and effector and also its central potentially economizing component, its structural-functional components open to strictly neurologic study and consideration, does not mean that all disorders must be explained as one has tried to explain aphasias. It is doubtful whether any specifically structural process or defect of the speech and language mechanisms should entail anything identical with or even comparable to the schizophrenic language disorders. The experiences with encephalitis make one limit the finality of such a statement.

Gruhle sees an unpleasant note in most of the delusions of schizophrenia—perhaps so, in a numerical and statistical sense. But why look on such states as those of ecstasy as merely a compensation for something unpleasant? Are they not due to the polarization, to the organization of so much of life in relativity, in opposites and contrasts, both ends of which have their right to recognition? Gruhle seems to scoff at the urge to disclose the reasons why any one trend becomes dominant. He seems to belittle Kretschmer's satisfaction with the intelligible character of the autistic love fulfilment in a meek reserved girl of 26. He seems happy when he can correlate some "secondary" delusions with some "primary" hallucinations, whereas most of us would probably try to understand the combination as a unit requiring an accounting without a preconceived formula. He does try, however, to tell us when and why certain delusions occur only in schizophrenia. The sample he uses (that of a patient who interpreted some dirt in the street as evidence that a boar had been led there to cause an allusion to his sexuality)

is strangely treated. He alludes to the mood theory of paranoia, and to the importance of the self-reference, the reference to his own sexuality as being without any adequate reason. "The paranoic fails to show even the slightest motivation or provocation." The patient is struck by something odd before there is any realization of any of the connections sensed later. Gruhle sees no evidence of complexes, desires or moods back of the delusion, but the real abnormality is the "delusion-function," not as yet tied to any specific content, and he makes this more acceptable by gloating over the failure of the discussion on the primacy of the intellect or the affects or the sensations or of Berze's apperception theory.

He is delighted with Neisser's term "morbid or faulty self-reference" in contrast to Meynert's delusion of being observed or Wernicke's "delusion of reference" and his "phenomenological" procedure; he refers to H. Schulte's original notion that man cannot exist long as a "Wir-Krüppel," a person deficient in belonging to a group. Gruhle seems to mention this only to say that this concept does not hold for the schizophrenic delusions (although Neisser discusses not paranoia alone but quite definitely the dementing delusional states). Gruhle thinks that, without any external and internal motives, the "process" originates the "experience of meanings, of symbols." One is here led into the old habit of psychologic quibbling due to the obsession of demanding a complete explanation of a vital phenomenon from a single integrative level, that of consciousness, the kind of thing Freud and others like to circumvent by hypothesizing the unconscious, the repression by resistance.

If one starts with the conviction that the delusion of schizophrenia is a process symptom, in contrast to that of the psychopathic paranoia, one can readily go on and claim that there are "no transition forms," even if, at the beginning, the two types may be "difficult to distinguish." Gruhle delights in dissecting with a gratuitous rationalization the naive freudian ebullitions on a case of paranoid jealousy, which must be a "Wahn-funktion" and schizophrenic. A belief or religious faith may really serve as a wish-fulfilment, but the paranoic person nearly always suffers seriously. He refers to double orientation in contrast to toxic disorientation, and to the false sense of recognition. He delights in "proofs" that the categories of normal psychology do not fit the pathologic case.

Gruhle shows the absence of self-reference in the delusions of dementia paralytica, the "concretization" of the delusions in the manic-depressive and the special types in the senile, but he cannot show a distinction in the delusional developments of epileptic patients. All depends on the distinction of delusion-content and delusion-function. One should contrast egocentric and nonegocentric delusions, perception-determined and free ones, those supported by hallucinations, those foreign to experience, the universal and the individualistic ones, the affectively marked and the affect-foreign notions, etc.

In the discussion of "the theory of schizophrenia," Gruhle comes closer to the essence, but he again loses himself in the multiplicity of actual formulas and the too detailed argument of the concepts of Bleuler, Berze and Kleist, treating the automatism theory of the French schools too secondarily. No one else experiences the automatism so strongly as foreign, induced, imposed and forced as does the consciously clear schizophrenic patient. The senile patient complains of a deficit, the melancholic patient of inhibition, the manic patient delights in his productions, the patient with dementia paralytica is uncritical, the toxic patient is too much kept out of that feeling or sentiment by the clouding of consciousness. Everywhere it is the relation to the self, the dispossession, the disappropriation, the nonannexation to the self, that makes the difference. Gruhle comes to the acknowledgment of many a good point in the French conception of Clérambault.

The "problem of the individual course" goes more definitely to the core of things. This is where his contrast between function and mere content problem becomes more effective. The function part must be common to all the schizophrenias, whereas it is a matter of pattern of content whether the patient should be persecuted by currents, by telepathy or by sexual incriminations, or whether he should have "all the five fundamental symptoms" together, whereas the

hebephrenic patient suffers largely from the schizophrenic basic mood and the schizophrenic inactivity. There may be a localization in the cortical regions or layers, or in the "nuclei." The distinctive developments are not merely a progression or activation of previous tendencies, such as a possible familial pattern (the specific combination being rarely the same in the same family).

One may well feel critically inclined toward this logical procedure. One needs a factual formulation which should as far as possible use all the facts, but without injecting assumptions that might close the doors of opportunity or search for opportunity.

Experience tells one to leave open all those items that really have to be settled by the course of events instead of being assumed as settled. It is better to start from facts (rather than words) which the rank and file of experience mark as demonstrated beyond dispute, or at any rate always open to retrial.

This is what has actually been done in the development of the formulation of dementia paralytica. With the too finalistic notion of the nineties, the pathologic anatomy in the hands of Nissl and Alzheimer and the clinical finalism of Kraepelin (emphasis on the outcome) created a sweepingly fatalistic formulation. Stanley Hall characterized the kraepelinian concept of dementia paralytica as "thanatic or deadly insanity." This included in the primary formulation something that should have been left to the course and not included in the initial assumptions or the "essence" of the "disease." A genetic-dynamic formulation would avoid including anticipations or tendencies as finished and final, closed facts. A static inclusion of merely possible tendencies as final and immutable neglects the biologic-plastic aspect of the facts on hand. In the case of dementia paralytica, Kraepelin assumed in 1896 an unknown cause, a metabolism disorder due to syphilis in most cases, but not directly syphilis of the brain, but rather a damage to the metabolism (the liver or what not?) that could also be brought on by other causes, such as alcohol. In this shyness of the directly syphilitic nature of the condition, he evidently was too cautious, to judge by later events, and he paid for lack of caution by making the "diagnosis" of dementia paralytica twice as often wrongly as correctly — a rather serious and humanly unforgivable side-effect of a presumably brilliant theory. Caution in behalf of a theory ultimately jeopardizing the patient is misplaced egocentric caution. The second serious slip consisted in Kraepelin's ignoring the interesting cases of "spontaneous" recovery which the later discoverer of the treatment with malaria, Wagner von Jauregg, kept before himself from the very beginning of his career, until the war period permitted a freedom of experimentation that led to success.

The fascination of the old nosologic urge to create absolutely pathognomonic signposts inevitably yields with difficulty to a principle that demands latitude and a readiness to pay attention to opportunity as well as fate and to admit occasionally the inability to see through unexpected developments. Man's thinking is based on principles of causality; but to accept a confession of modesty with respect to one's sagacity and an admission of one's not being wholly omniscient seems to be difficult in a civilization based on competition and eternal comparison. Hence the willingness to borrow dignity by accepting a faith and creed, or to disclaim all dependability and responsibility in rank opportunism, and a relatively small ratio of efforts to steer between the extremes in a melioristic determination and sense of personal standards, somewhat after the doctrine recommended by Arnold Bennett, viz., to treat everybody else as a machine and oneself as a responsible creative agent.

The experience with Kraepelin's work with "disease entities" and dogmatic preoccupations should keep one from doing the same thing in the most important and most frequent mental disorder that today fills our state institutions. 1. It is presumptuous to operate with a concept that tries to settle prematurely by merely collateral evidence the justifiability of a basic assumption. As long as the course and the origin are uncertain and the structural facts are as debatable as they are, one must not introduce prejudicial entities and delimitations into the scheme. 2. It is therefore wiser to keep the door open to the facts as found and viewed for

what they may be worth in the light of experimental formulation and scrutiny by actual test rather than legitimation largely by logic.

The monograph here reviewed contains much evidence of hard work, a wealth of experience and a noteworthy level of erudition. One might be justified in feeling aghast, however, at the sterility as to any benefit to the patient or a more effective understanding of the conditions discussed. One feels a struggle for academic standing and comprehensiveness, admirable if it leaves the door open to a real interest in a creative experimentalism, to a willingness to see the facts as well as one can today and a preference for what one can control practically and experimentally. If one could attack the problem from a structural or physiologic end, by intercepting the evil work of poisons, by the avoidance of mishaps through negative and positive eugenics, or by the striving for fewer discrepancies in the individual development, one might have less cause for literary pride, but probably a more open mind for the use of the facts that one knows. There is sense in the study of the full-fledged cases when they teach one to pay attention to their evolution and to work with the component factors before they have reached the stage for nothing but diagnostic pride.

THE BRAIN FROM APE TO MAN. A CONTRIBUTION TO THE STUDY OF THE EVOLUTION AND DEVELOPMENT OF THE HUMAN BRAIN. By FREDERICK TILNEY, PH.D., M.D. With Chapters on the Reconstruction of the Gray Matter in the Primate Brain Stem by Henry Alsop Riley, A.M., M.D., and Foreword by Henry Fairfield Osborn, Sc.D., LL.D. Price, $25. Vol. I., pp. 473; Vol. II., pp. 1045. New York: Paul B. Hoeber, Inc., 1928.

These volumes, impressive less in their size than in their contents, represent a neuro-anatomist's scholarly attempt to arrange all of the evidence that neuro-anatomy can present on the subject of the evolution of the human brain. According to professor Osborn, "This volume contains the basis of what to our knowledge is the first profound study of the genesis of the intimate and internal structure of the human brain in comparison with the brains of animals more or less nearly related to man . . . . yielding a result of exceptional breadth, precision and exactitude."

To describe the book merely as an exhaustive anatomic study — which it undoubtedly is — would, however, be inadequate. Besides being a neuro-anatomist of the first rank, the author is a clinician and a trained observer of the behavior of man and other animals. All of these points of view he has brought into the making of this book, weaving the one into the other until the reader at first wonders whether he has read a textbook of comparative anatomy, a treatise on behavioristic psychology or a study of the evolution of man. Further reflection tells him that he has read all three, blended into a perfect story of the brain from ape to man, in all that the brain means in mammalian life.

Dr. Tilney works with a massive material, morphologic and observational, and arranges it in a steplike series that makes his story clear and leaves "no room to doubt that a progressive functional development has gone hand in hand with impressive evolutional modifications in the structure of the brain." The material is arranged in four groups: I. The Lower Primates; II. The Intermediate Primates; III. The Higher Anthropoids, and IV. Man. Among the lower primates he places: *Lemur mongoz, Tarsius spectrum, Callithrix jacchus* and *Mycetes seniculus.* The intermediate primates consist of: *Papio cynocephalus, Pithecus rhesus* and *Hylobates hoolock.* The higher anthropoids are: *Simia satyrus* (the orang-outang), *Troglodytes niger* (the chimpanzee) and *Troglodytes gorilla.*

Each form is considered separately in the order given and an orderly routine is followed in each description. First one is presented with a description of the animal, including his habitat and what is known of his history, his habits of life and activities both in the wild state and under domestication. Special stress is laid in each form on the characteristics of the hand and foot, for the author tells a story of these members which illuminates his theory of the close relationship

between their development and the development of the forebrain. Then the brain is presented — the gross anatomy of the external appearance and of the brain stem. More importance still is given to the internal structure of the brain stem which is demonstrated by cross-sections of the stem — from ten to sixteen for each animal — running from the level of the pyramidal decussation up into the thalamus. These sections are carefully chosen from nearly identical levels in each brain so that easy comparison is possible. The illustrations are clear halftones, on one half of which the different structures are outlined and labeled. The accompanying text is complete, concise and easy to follow. After each of these studies comes Dr. Riley's chapter on the reconstruction of the gray matter of the brain stem, with cuts in color and in outline and a clear descriptive text. This anatomic part alone is an accurate and authentic scientific work, complete enough to be used by students as a text or reference book. At the end of each of his main groups the author presents a chapter of summary in which he evaluates his evidence and arranges it in orderly sequence to tell his story of the evolution of the brain, which has gone hand in hand with the evolution of intelligence.

The second and larger volume deals with the higher anthropoids and man. The same orderly routine is followed, but one finds more of the habits and activities of the animals. These descriptions, partly original observations by the author and partly from other authentic sources, are entertaining and instructive, forming a delightful basis on which the brain structures may be understood.

The section on man brings the story to its climax and one sees the reason for all the description that has gone before. Man is studied from the start to bring his developmental history up to modern times. Beginning with *Pithecanthropus* and *Eoanthropus*, the author leads us up to *Cromagnon* man, telling in charming fashion the story of man's development in a concise abstract of what modern anthropology can teach of the different known forms of man as revealed by the study of fossils and of the instruments and tools he has left behind.

Following the description of the human brain from gross appearance, cross-section and reconstruction, there comes an interesting attempt to evaluate the life activities of primitive man in relationship to the size and form of his brain. This is accomplished by a study of the brain casts of many primitive skulls, profusely illustrated and adequately described. These chapters are unique; the reviewer knows of no other work that contains all of this material so completely and so concisely arranged.

In the concluding chapter the author tells anew in rapid narrative his story of the evolution of the brain and the evolution of intelligence, bringing all his evidence together summarily with new illustrations and new material the further to prove his points.

A final word of appreciation must be said of this book, not as a scientific treatise but as a book. It is a readable story, accurate in its details, fascinating in its scope and delightful in its presentation. Dr. Tilney has brought to this monumental task, besides the enthusiasm of an anatomist, a well controlled imaginative vision, a clarity of diction and a charm of literary style that does credit not only to American science but to American letters.

LES PSEUDOBULBAIRES. By ROBERT THUREL. Price, 40 francs. Pp. 362. Paris: Gaston Doin, 1929.

This study from the service and laboratory of the Salpêtrière consists in a magnificent collection of 100 cases of pseudobulbar palsy, with a detailed study based not only on this, but on the preceding literature. It follows the usual exposition of a syndrome in taking up the characteristic and then the variable features, of which there is such an abundance in pseudobulbar palsy. It represents indeed an intensive study, which is better on the clinical than on the pathologic side. The conditions giving rise to the syndrome are numerous, including arteriosclerosis, syphilis, multiple sclerosis, epidemic encephalitis, cerebrospinal meningitis and occasionally intoxications, tumors and traumatisms. The section on involuntary laugh-

ing and crying is particularly good, although it lacks the art of exposition of Wilson's paper.

The author's study of the development of pseudobulbar palsy is important, since he states that it comes on "progressively, without ictus, preceded a long time by certain accessory symptoms which should arrest the attention: hyperemotionalism, irritability, imperious micturition; which are indicative of disseminated lesions." Moreover, in numerous cases the strokes are incidental phenomena and are not really the first manfestations of cerebral sclerosis.

The bibliography is unusually full. The work is nicely printed, and the illustrations are satisfactory.

DIE HYPOPHYSE. VORTRÄGE GEHALTEN IN DER WIENER GESELLSCHAFT FÜR INNERE MEDIZIN UND KINDERHEILKUNDE. By A. EISELSBERG, O. MARBURG, M. SGALITZER, O. HIRSCH, J. NOVAK and E. A. SPIEGEL. Price, 3.50 marks. Pp. 66. Berlin: Urban & Schwarzenberg, 1930.

This small monograph consists of a series of lectures delivered by Eiselsberg, Marburg, Sgalitzer, Hirsch, Novak and Spiegel. A lecture delivered by Erdheim is not included because he has covered the same ground in other publications. The subjects covered include the anatomy and physiology of the hypophysis, the clinical manifestations of hypophyseal tumors, the operability of hypophyseal neoplasms, the roentgen treatment for these tumors and a study on the relation between the hypophysis and the genitals.

Spiegel's lecture on the anatomy and physiology of the hypophysis is a useful review. Sgalitzen's lecture on the roentgen treatment for tumors of the hypophysis is of interest. In five cases of acromegaly, two were markedly improved by roentgen treatment, one remained stationary, and two were refractory to treatment. Of ten cases of lipodystrophic disorder, the symptoms of the eyes which were severe at the time of treatment in six cases were much improved, while three remained unaffected and one stationary. He believes that roentgen therapy is helpful in the treatment for the disorders of the eyes, and states that in cases of this sort which do not react to the roentgen rays the patient must be operated on. The pamphlet constitutes a useful compendium.

THE STRUCTURE AND MEANING OF PSYCHOANALYSIS AS RELATED TO PERSONALITY AND BEHAVIOR. By WILLIAM HEALY, M.D., A. F. BRONNER, PH.D., and A. M. BOWERS, A.B. Price, $5. Pp. 482. New York: Alfred A. Knopf, 1930.

In attempting a thorough general review of the literature for the purpose of showing the influence of psychoanalysis as one of the scientific approaches to understanding of personality, the authors found it rather difficult to present the subject in a comprehensive and efficient manner and this book is the result. It is an excellent presentation of the whole system of discoveries, technic and theories of psychoanalysis. The value of the work consists in the completeness of its presentation and in the fact that it gives a logical orderliness to psychoanalytic development and thought. A rather unusual method of presentation is utilized. On the left hand pages the basic concepts, chiefly of Freud and his followers, are presented. On the right hand are the elaborations, corroborations and divergencies. There are no comments. The book will serve very well as an introduction to the study of psychoanalysis.

DIFFERENTIALDIAGNOSTIK IN DER PSYCHIATRIE. By HERMANN HAYMANN. Mit einem Beitrag: Die diagnostische Bedeutung der Intelligenzprüfungen, von Erich Stern. Price, 17 marks. Pp. 261. Dresden: Theodor Steinkopff, 1930.

In a concise presentation, Haymann presents an orthodox discussion of symptomatology and nosology current in the German texts. In no field as much as in

psychiatry, are diagnosis, understanding and treatment so strongly interlaced that one wonders how they could be separated to advantage. As part of a handbook of differential diagnosis, this volume evidently had to follow principles more easily made effective in other domains of medicine.

The division written by Erich Stern gives an interesting picture of the utilization and evaluation of tests in German practice, from an interesting group of tests on infants (Hetzer and Wolf, covering the first year) to vocational guidance and an effort to draw a line between essentially pedagogic and medical practice.

SLEEP AND THE TREATMENT OF ITS DISORDERS. By R. D. GILLESPIE. Price, $3.25. Pp. 267. New York: William Wood & Company, 1930.

The practicing neurologist is consulted perhaps more frequently by the patient because of insomnia than for any other symptom. This book, therefore, which is primarily designed for the practitioner, should be of great use, for, besides giving an excellent résumé of the present point of view of the physiology and pathophysiology of sleep and the effects of sleeplessness, it presents a sane view of the treatment to be adopted in such conditions. The use of drugs is fully discussed.

GREAT EXPERIMENTS IN PSYCHOLOGY. By HENRY E. GARRETT, PH.D. Price, $2.50. Pp. 330. New York: Century Company, 1930.

This book is obviously designed for the beginner in psychology. Its aim is to give the student some idea of how psychologic facts have been discovered and to familiarize him with some of the leaders who have contributed to its advancement. It fulfils its purpose admirably.